ENDANGERMENT ASSESSMENT
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

9-1-86

PROJECT FOR PERFORMANCE OF REMEDIAL RESPONSE ACTIVITIES AT UNCONTROLLED HAZARDOUS SUBSTANCE FACILITIES—ZONE 1

NUS CORPORATION
SUPERFUND DIVISION

LEE 001

002070

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Contents

Tables

Figures

Appendices

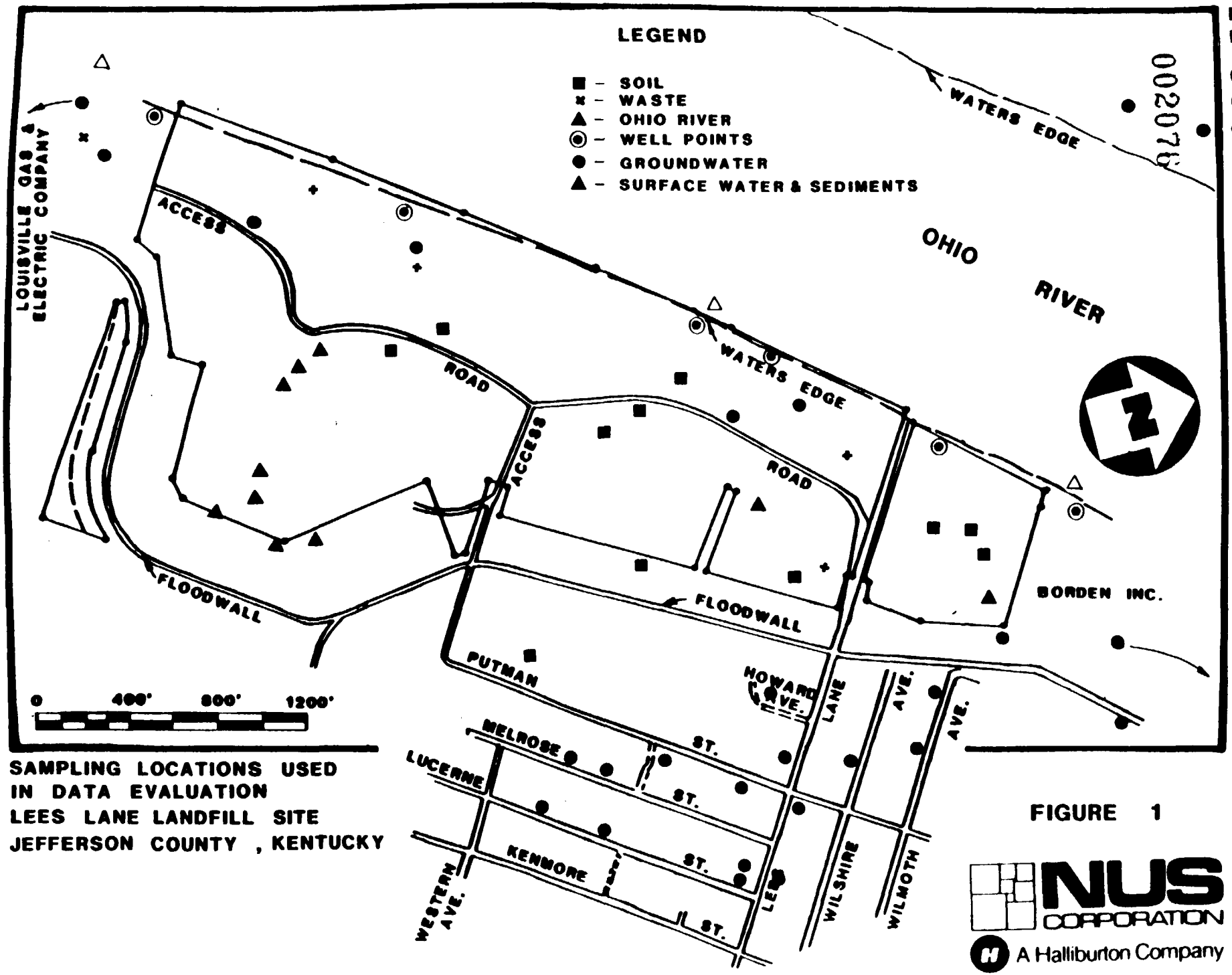
1.0 INTRODUCTION

A Remedial Investigation (RI) was performed at the Lees Lane Landfill Site by the NUS Corporation in order to characterize the types and extent of contamination. The purpose of the RI was to compile sufficient data to identify the contaminants of concern, determine potential public health and environmental problems and support the development and evaluation of remedial alternatives during the Feasibility Study (FS).

The RI included the installation and sampling of five new monitor wells and the sampling of existing onsite monitor wells, residential, industrial and public supply wells, surface soil, sediment and water, and the Ohio River. Figure 1 shows all of the sample locations for data used during the RI. An FS was also performed by the NUS Corporation to assist the EPA in selecting the appropriate remedial action alternative for the Lees Lane Landfill Site. The purpose of the FS was to identify and evaluate remedial alternatives with a range of responses from no-action to offsite disposal and/or treatment. The evaluation of remedial alternatives was based on technological, public health, institutional, environmental, and cost factors. The final RI/FS was submitted to EPA in April 1986.

The purpose of this Endangerment Assessment is to evaluate the actual or potential harm to the public health, welfare, or the environment presented or potentially caused by the threatened or actual release of hazardous substances. In general the endangerment assessment will characterize (1) the nature and distribution of contaminants present, (2) the environmental fate and transport mechanism within specified environmental medium (3) intrinsic toxicological properties of specified substances, (4) exposure pathways, (5) populations at risk and (6) the potential health risks associated with the presence of hazardous substances at the site.

This Endangerment Assessment will not provide an in-depth evaluation of the air media due to insufficient data available. The air investigation is currently being implemented at and in the vicinity of the site. After all data have been collected and evaluated, this data will be available as an addendum to the Endangerment



SAMPLING LOCATIONS USED
IN DATA EVALUATION
LEES LANE LANDFILL SITE
JEFFERSON COUNTY , KENTUCKY

FIGURE 1

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Assessment. The information for this Endangerment Assessment was taken from the April '86 RI/FS for the Lees Lane Landfill Site.

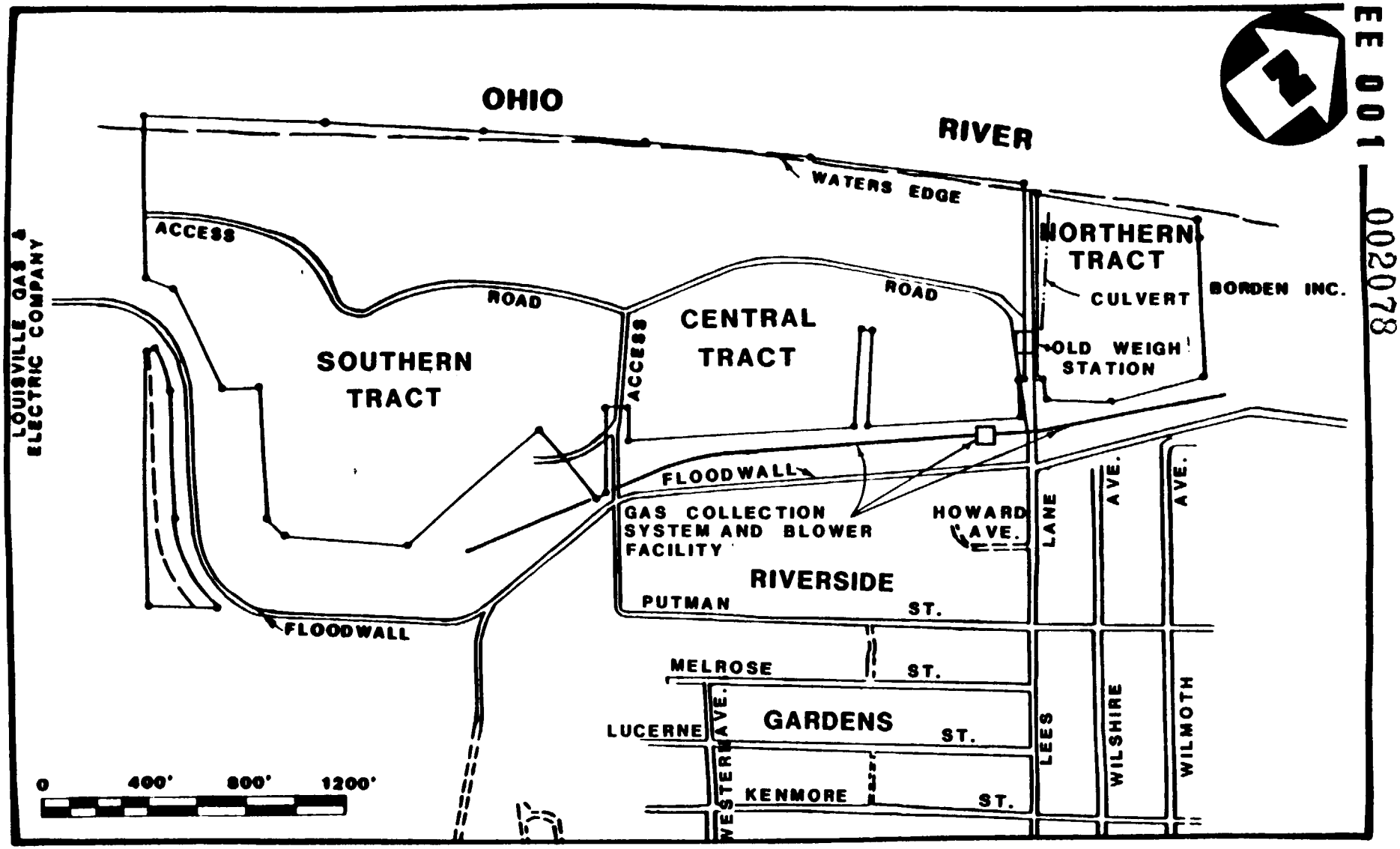
1.1 Site Description and History

The Lees Lane Landfill Site is located adjacent to the Ohio River in Jefferson County, approximately 4.4 miles southwest of Louisville, Kentucky. The site, consisting of 112 acres, is composed of three tracts and measures approximately 5,000 feet in length and 1,500 feet in width (see Figure 2). The northern and central tracts of the landfill consist of level to gently sloping land while the southern tract contains two depressions with steep slopes. Up to three terraces, each approximately 20 feet wide, form the slope on the river side of the landfill. Much of the landfill surface is covered with well-established vegetation ranging from brush to woodlands. Elevations range from 383 feet above mean sea level (amsl) along the Ohio River to 461 feet amsl along the levee.

The site is bordered on the east and south by a flood protection levee (designed on the 500-year flood). To the northeast is Borden, Incorporated (a chemical manufacturer), to the south is Louisville Gas and Electric, Cane Run Plant (a coal-burning electricity generating station), and to the east is Riverside Gardens (a residential development of about 330 homes and 1,100 people). Beyond these areas the surrounding land use is predominantly woodlands and agricultural land (see Figure 3).

The Lees Lane Landfill Site is located in the glacial outwash and alluvium of the Ohio River. Sand and gravel were quarried at the site as early as the 1940s and the excavated areas were probably landfilled concurrently with the quarrying operations.

The site is underlain by an alluvial aquifer extending to the shale bedrock approximately 110 feet below the land surface. The saturated thickness of the alluvial aquifer is approximately 60 feet, allowing a 50-foot buffer between the normal groundwater levels and the land surface. The depth of excavation at the



SITE LAYOUT

LEES LANE LANDFILL SITE

JEFFERSON COUNTY, KENTUCKY

FIGURE 2

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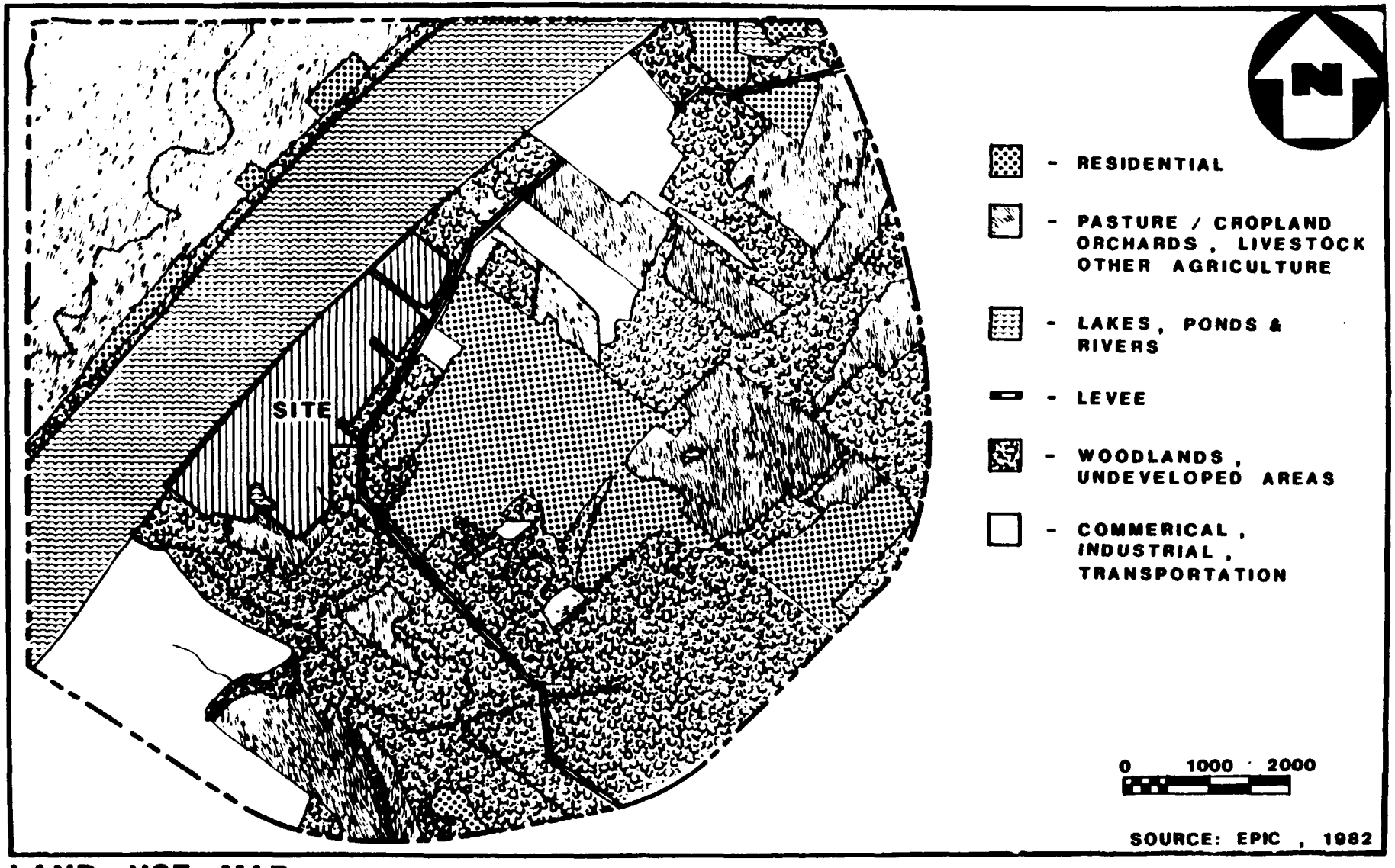

LAND USE MAP
LEES LANE LANDFILL SITE
JEFFERSON COUNTY , KENTUCKY

FIGURE 3

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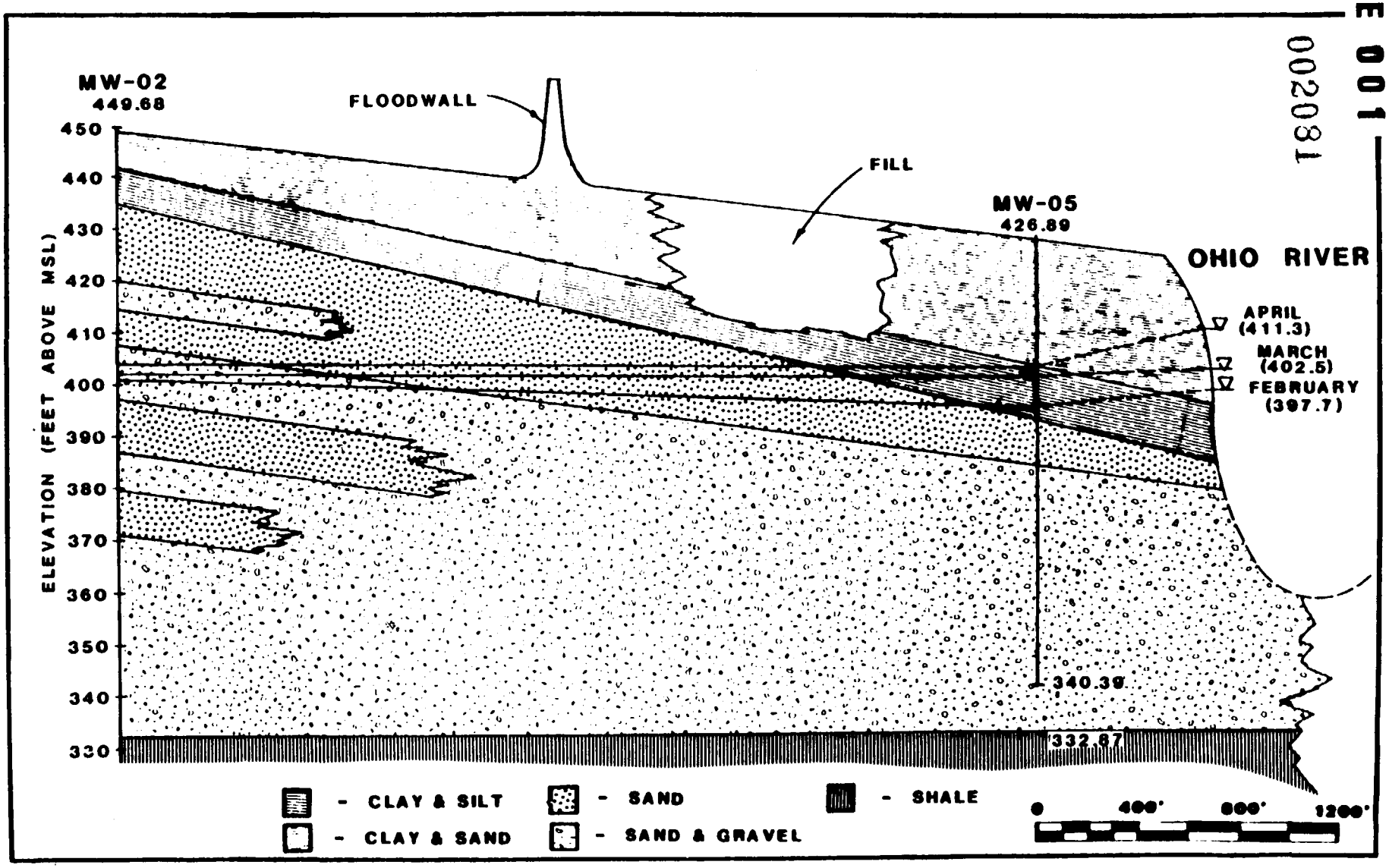
site is unknown but most of the sand and gravel pits are not expected to have exceeded 25 feet. Figure 4 depicts the generalized geological conditions at the site.

The landfill was closed in 1975 and the fill appears to have been covered with local soil materials. The site was not graded to promote drainage and currently the landfill surface is irregular with a large depression located in the southern tract, where capacity remained when the landfill was closed. The cover material appears to be relatively permeable based on the lack of standing water observed during the conduct of the RI.

The permeable cover material cannot be expected to inhibit infiltration of rainwater and subsequent leachate production. There is no evidence of the use of landfill liners or leachate collection systems to prohibit the migration of leachate to groundwater. It does not appear that the landfill has stabilized based on the observation of undulations in the access road probably caused by the compaction of wastes within the fill.

The volume of fill at the site has been estimated at 2.4×10^6 cubic yards, but little information is available as to the actual composition of the wastes. Municipal, industrial, and commercial wastes are known to have been disposed of in the landfill but no records exist as to the type or location of specific wastes. Based on a Congressional Survey conducted in 1979, both hazardous and nonhazardous wastes have been placed in the landfill and landfill practices at the time of operation suggest that these wastes were probably comingled within the various pits.

Access to the landfill is currently uncontrolled and recreational use is evident. Hunters and fishermen were observed regularly during the conduct of the RI. In addition, indiscriminant dumping on the landfill surface can be observed along the access road at various locations throughout the site.



CROSS-SECTION MW-02 - MW-05

LEES LANE LANDFILL SITE

JEFFERSON COUNTY, KENTUCKY

FIGURE 4

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1.2 Basis of the Endangerment Assessment

In general, the Endangerment Assessment describes the hazardous compounds of greatest concern, assesses the significant contaminant migration routes and exposure pathways that have been identified, and evaluates potential adverse effects to the susceptible receptors. Several factors were considered during the implementation of this assessment. They include:

- Present site conditions pertinent to the selection of critical contaminants, as defined by the RI
- Physical, chemical, and biological characteristics affecting the environmental fate and mobility of the contaminants
- Health effects and the environmental impacts associated with exposure to the contaminants, including additive, synergistic, or antagonistic effects.

Some limitations affected the extent these factors could be evaluated and therefore constrained the scope of the assessment and the conclusions that could be inferred. The limitations included the quality of the laboratory analytical data, availability of toxicological data on the contaminants present, relevance of toxicological data to site-specific exposure scenarios, and the degree to which probabilities of exposure could be estimated or predicted. Although some limitations did exist, they were not severe and did not prevent the development of a selection of critical contaminants.

1.3 Selection of Critical Contaminants

Hazardous gases and methane have the potential for subsurface migration to the Riverside Gardens area; however, the operation of the existing gas collection system between the landfill and the residences appears to have temporarily mitigated the problem. Potential inhalation of hazardous gases could also pose problems to nearby residents; and a separate air investigation is being conducted at

and in the vicinity of the site. Therefore, the Endangerment Assessment presented here will not consider contaminants in the air media.

A number of inorganic and organic contaminants were detected in the other media at the Lees Lane Landfill Site. The majority of the contaminants observed have relatively low toxicities or were found infrequently and were not considered typical of the site conditions in general. A select few, however, were widespread at the site or had important chemical or biological toxicological properties, and were considered contaminants of concern.

Receptors could be exposed to low-level contaminants in the surface water, sediment, or soil through direct contact. However, nearby private wells in Riverside Gardens and the Edwardsville Water Company public water supply wells withdraw groundwater from the alluvial aquifer which is beneath the site. Therefore the selection of critical contaminants has been based on the contaminants detected in the groundwater system.

Each of the contaminants and the maximum concentration detected in groundwater at or near the site is presented in Table 1. Significant differences in concentrations, frequency of detection, and the physical, chemical, and biological characteristics of these contaminants are evident. But an evaluation of the characteristics for each contaminant is not necessary for the completion of an Endangerment Assessment. A detailed examination of a set of major contaminants will provide adequate input for the Feasibility Study (FS), and will result in a representative analysis of existing site conditions.

The following criteria were used to select or exclude the critical contaminants:

- Tentatively identified compounds were not considered. Contaminants omitted for this reason include: hexahydroazepinone, pentanoic acid, butanoic acid, octanoic acid, hexanoic acid, tetradecanoic acid, ethylhexanoic acid, benzene acetic acid,

TABLE 1
MAXIMUM CONCENTRATION OF CONTAMINANTS IN GROUNDWATER
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

Parameter	Concentration (ug/l)
Extractable Organics	
Di-N-Butyl Phthalate	8
Bis(2-Ethylhexyl)Phthalate	20J
Phenol	300J
4-Methyl Phenol	2J
Methylpentanoic Acid	6J
Pentanoic Acid	7JN
Butenoic Acid	10JN
Octanoic Acid	9JN
Hexanoic Acid	10JN
Dodecanoic Acid	20JN
Hexadecanoic Acid	10JN
Tetradecanoic Acid	5JN
Benzoic Acid	2J
Ethylhexanoic Acid	10JN
Benzeneacetic Acid	10JN
Hexahydroazepinone	50JN
Purgeable Organics	
Chloroform	5J
Benzene	450
Toluene	10J
Propanol	200JN
2-Propanol	9,000JN
Ethanol	30JN
Butanol	10JN
Butanoic Acid, Ethyl Ester	50JN
Butanoic Acid, Butyl Ester	20JN
Methyl Ethyl Ketone	30J
Dichlorofluoromethane	6JN
Methyldioxalane	5JN
Diethylether	40JN
Carbon Disulfide	49
Inorganics	
Silver	15
Arsenic	87
Barium	1,100
Cadmium	22
Cobalt	160
Chromium	640

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TABLE 1
MAXIMUM CONCENTRATION OF CONTAMINANTS IN GROUNDWATER
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY
PAGE TWO

Parameter	Concentration (ug/l)
Inorganics (cont'd)	
Copper	220
Nickel	340
Lead	150
Vanadium	270
Zinc	3,200
Aluminum	85,000
Manganese	7,900
Calcium	350,000
Magnesium	150,000
Iron	190,000
Sodium	63,000
Cyanide	20J
Potassium	61,000
Chloride	63,000
Sulfate	630,000

J Estimated value
N Presumptive evidence of presence of material

propanol, 2-propanol, ethanol, butanol, butanoic acid (ethyl ester), butanoic acid (butyl ester), dichlorofluoromethane, methyldioxalane, and diethylether.

- A contaminant was eliminated from consideration as a critical contaminant if it were found infrequently so that it was estimated to not be widely enough distributed to result in a potential health risk. Contaminants eliminated by this criterion are: phenol, 4-methyl phenol, toluene, carbon disulfide, silver, cadmium, cobalt, vanadium, and cyanide.

- If there were significant health consequences based on toxicological potential or level of exposure associated with an individual contaminant in site-specific circumstances, it was considered significant and included in the assessment. Conversely, if the opposite were true, the contaminant was not considered. Contaminants omitted for this reason include: di-n-butylphthalate, bis(2-ethylhexyl)phthalate, methylpentanoic acid, benzoic acid, methyl ethyl ketone, barium, copper, nickel, zinc, aluminum, manganese, calcium, magnesium, iron, sodium, potassium, chloride, and sulfate.

Iron and manganese were frequently detected in elevated levels in groundwater in the vicinity of the site. Since these elevated levels were widespread and detected in background samples, the problem appears to be areawide and not specifically related to the site. Although the concentrations of iron and manganese exceeded the maximum contaminant levels (MCLs) set by the National Secondary Drinking Water Regulations in a number of onsite monitoring wells and offsite public drinking water wells, these two contaminants will not be addressed in detail since they have low mammalian toxicities. Toxic effects associated with iron and manganese generally only occur from occupational exposure to dusts and fumes and adverse effects have not been reported from oral ingestion in man or animals. The main reason for limiting iron and manganese concentrations in drinking water is for aesthetic reasons and to prevent objectionable tastes.

As a result of the application of these selection criteria, only four contaminants remained. These contaminants are considered critical contaminants and potential health effects related to exposure to them was more thoroughly examined.

Table 2 provides a summary of the range of concentrations of the critical contaminants found in the various media at the Lees Lane Landfill Site. Table 3 provides the concentrations of the critical contaminants in the shallow portion of the aquifer, while Table 4 provides the concentrations in the deeper portion of the aquifer.

2.0 ENVIRONMENTAL TRANSPORT MECHANISMS

The transport of site contaminants is dictated by the release mechanism for the contaminant, the migration route, and the the applicable mitigating factors associated with each environmental media. These transport mechanisms are summarized in Table 5 and described in detail by environmental media.

2.1. Groundwater

The source of contaminants to groundwater is the result of leachate migration from the landfill. Continued leachate production is expected due to the permeable landfill cover. Containment of leachate is not expected since there are no known landfill liners or leachate collection systems in use at the site. Downward percolation of leachate through the natural alluvial and glacial outwash materials surrounding the fill is not expected to provide any measurable attenuation of contaminants.

The groundwater flow direction at the site is predominantly toward the Ohio River with discharge into the River (see Figure 5). However, based on 60 feet of saturated thickness of the aquifer and up to 30 feet from the bed of the Ohio River to the shale bedrock below, there is a potential for contaminants to travel under the Ohio River and into Indiana. During periods of high flow in the Ohio River, contaminant migration may reverse. If this occurs, there is a potential for

TABLE 2
CRITICAL CONTAMINANT LEVELS IN VARIOUS MEDIA
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

Critical Contaminant	Groundwater (ug/l)	Surface Water (ug/l)	Bottom Sediments (mg/kg)	Surface Soil (mg/kg)
Lead	ND-150	ND-10J	10J-100J	50J-2,000J
Arsenic	ND-87	ND	5.4-27	ND-25
Benzene	ND-450	ND-5J	ND-15J	ND
Chromium	ND-640	ND-6.2	9.8-30J	10J-2,000J

ND Not detected.

J - Estimated value.

TABLE 3
DISTRIBUTION OF CRITICAL CONTAMINANTS IN GROUNDWATER
TOP OF AQUIFER
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

Critical Contaminant in ug/l	Upgradient Shallow Well 12/84	Onsite Shallow Wells 12/84	Well Points at Ohio River 11/84
Lead	7.2	ND - 150	17 - 83
Arsenic	-	ND - 87	ND - 60J
Benzene	-	ND0	ND- 450
Chromium	43	12 - 140	ND- 33

ND Not detected.

J Estimated value.

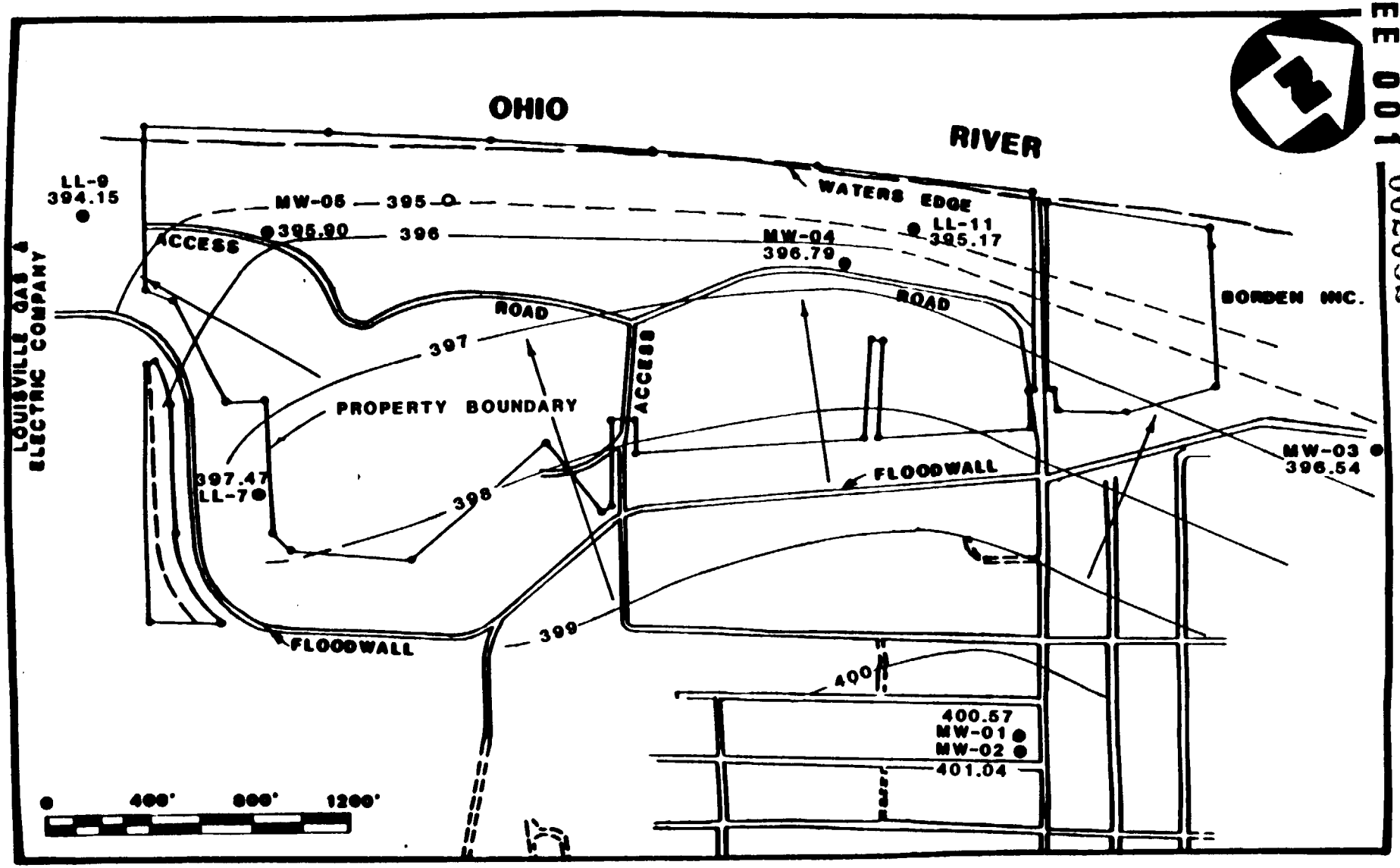
TABLE 4
DISTRIBUTION OF CRITICAL CONTAMINANTS IN GROUNDWATER
BOTTOM OF AQUIFER
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

	Upgradient		Northern Tract East of Fill		Central Tract Onsite		Southern Tract Onsite		Downgradient Indiana Public Supply Wells		Industrial Wells
Contaminant in ug/l	12/84	1/85	12/84	12/84	1/85	12/84	1/85	12/84	1/85	12/84	12/84
Lead	28	23J	20	68J	15	17J	11	44J	10	2.8J	-
Arsenic	ND	ND	ND	4.3	-	7.3	-	8.1	-	-	-
Benzene	ND	20	ND	ND	-	-	-	-	-	-	-
Chromium	120	57J	640	210	230	30J	360	400	12	R	-

ND Not detected.
J Estimated value.
R Quality Control indicates data are unuseable.

TABLE 5
CONTAMINANT TRANSPORT MECHANISMS
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

Affected Area	Affected Media	Release Mechanism	Migration Route	Mitigating Factors
Offsite	Groundwater	Leachate	Into Ohio River Under Ohio River Into Riverside Gardens	Dilution Dilution Infrequent
	Surface Water Sediments Surface Soil	Runoff	Into Ohio River	Infrequent/Dilution
	Gas Migration	Excavation	Into Riverside Gardens	Collection System
	Air	Gas Production	Into Riverside Gardens	Dilution
Onsite	Surface Water Sediments Surface Soil Air	Unrestricted Access	To Pond in S. Tract To Pond in S. Tract To Pond in S. Tract Thru Cover Material	Infrequent Use



GROUNDWATER CONTOURS
DECEMBER 4 , 5 , & 8 , 1984
LEES LANE LANDFILL SITE
JEFFERSON COUNTY , KENTUCKY

FIGURE 5

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transport of landfill contaminants into Riverside Gardens. In addition, past operation of a pumping center to the northeast of the site may have diverted groundwater flow. A careful evaluation of each of these migration routes was made as part of the RI. Continuous water level recorders were placed on two monitor wells and groundwater quality samples were collected throughout the site.

Under normal flow conditions in the Ohio River, most of the potentially contaminated groundwater can be expected to discharge to the River. Based on conservative estimates, the discharge to the Ohio River will not exceed 1.69 cubic feet per second (cfs). The average flow of the Ohio River at Louisville is 114,000 cfs; and therefore, the groundwater contribution is 0.0015 percent of the total flow.

Flow under the Ohio River is not expected to occur to any large extent based on the relatively flat bedrock beneath the site (dipping 8.3 feet per mile). Areas of high relief located less than a mile from the Indiana bank of the Ohio River may contribute to a steeper groundwater gradient in Indiana than groundwater at the site (the maximum observed gradient during the RI was 0.007). If flow under the Ohio River were to occur, some dilution from the River would be expected as the waters comingled beneath the River.

Continuous water level recorders placed on the monitor wells during the RI indicated a rapid groundwater response near the River in the central tract to rises in Ohio River stage. Little response to the pumping center to the northeast of the site was observed.

The potential for flow reversal of shallow groundwater within the landfill boundaries was observed during the RI. During periods when the Ohio River water levels were high (greater than 400 feet above mean sea level (amsl)), the monitor well near the river in the central tract exhibited a water level at a higher elevation than the monitor well east of the northern tract. Extensive groundwater flow reversal conditions have not been established at the site. In order for groundwater flow reversal to reach Riverside Gardens, the conditions necessary for groundwater

flow reversal would have to be present for a long period of time. this is extremely unlikely; and therefore, flow reversal to Riverside Gardens is also very unlikely.

Onsite groundwater contained low levels of organic compounds and some inorganic contaminants. The major inorganic contaminants included arsenic (87 ug/l), barium (1,100 ug/l), cadmium (22 ug/l), chromium (640 ug/l), lead (150 ug/l), manganese (44,000 ug/l), and iron (190,000 ug/l). The offsite concentrations of these contaminants were all below the maximum contaminant levels (MCL) set in the Interim Primary Drinking Water Regulations. Manganese was detected at 610 ug/l in the Louisville Gas and Electric well and at 370 ug/l in an Indiana public water supply (PWS) well. Iron was detected at 8,900 ug/l in an Indiana PWS well, but was below background in both industrial wells. Neither manganese or iron are considered to have significant health effects.

Very little potential exists for the migration of landfill contaminants into Riverside Gardens. Samples from residential wells were collected in 1978 and again as part of the RI. The distribution of contaminants within the groundwater samples is sporadic and does not suggest migration beyond the landfill boundary toward Riverside Gardens.

The flow rate of groundwater was calculated to be 420 feet per year using nonconservative parameters. Based on an approximate width of the landfill of 1,500 feet, it can be expected that groundwater entering the site at the upgradient boundary would travel approximately 3.6 years beneath the landfill before discharging to the Ohio River. Accounting for variations in gradient due to higher water levels in the Ohio River, this flow rate should not be expected to be more than double.

2.2 Surface Water

The potential sources of contamination for surface water at the site include runoff over contaminated surface soils, leachate seeps and groundwater discharge. Based on the permeability of the cover material, most runoff is expected to infiltrate the

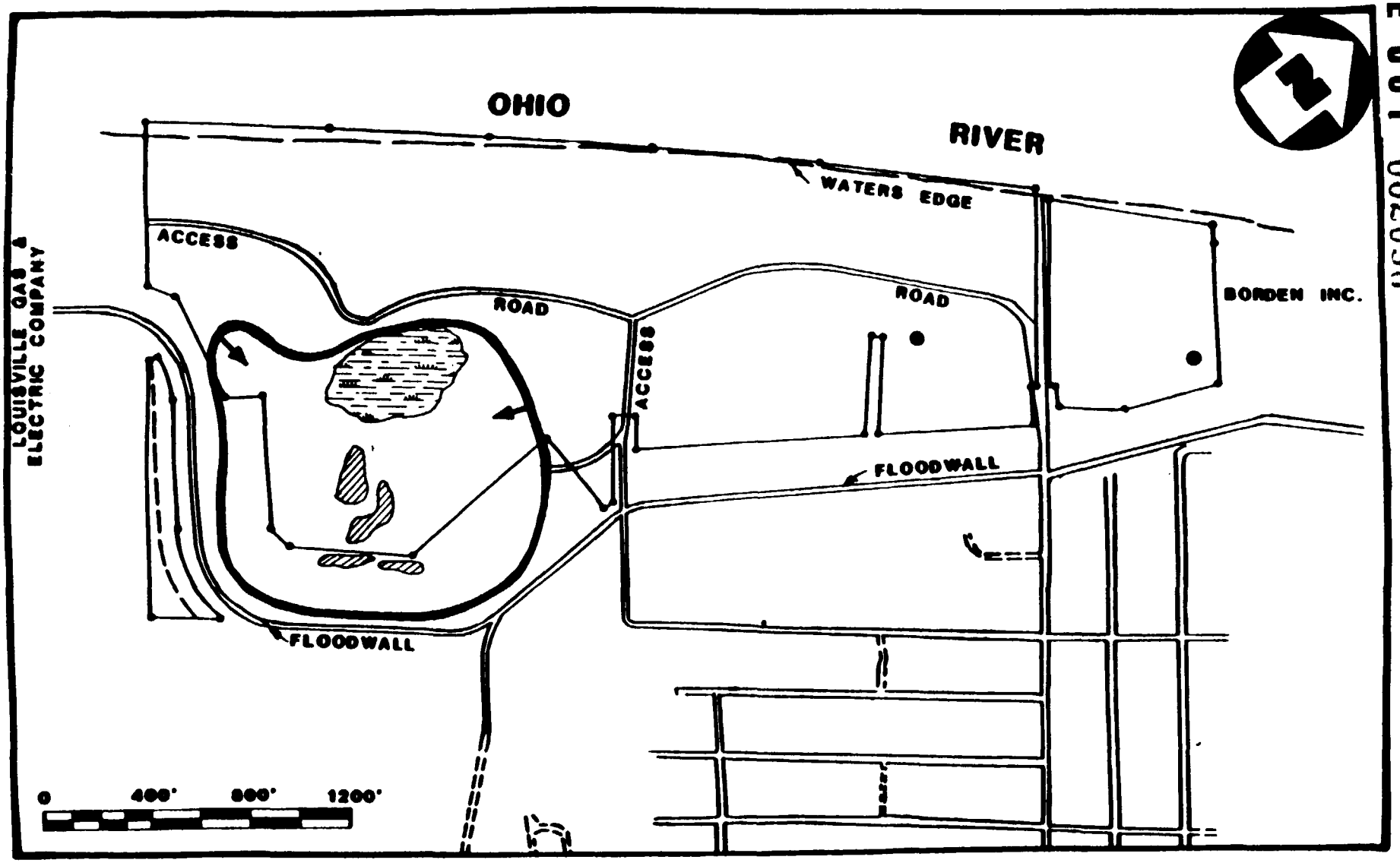
fill in the northern and central tracts rather than to stand on the landfill surface. The topography of the southern tract suggests that some runoff is likely to accumulate in the pond formed by a depression resulting from incomplete filling (see Figure 6).

The floodwall/levee at the site extends along two sides of the landfill boundary, prohibiting runon of surface waters. In addition, a topographic high on the northeast side of the landfill tends to inhibit runon in this area. Therefore, little runon occurs at the site and most of the runoff leaving the site is discharged directly to the Ohio River. A small amount of runoff enters Mill Creek Cutoff near the Ohio River, but it is expected to be transported immediately to the Ohio River.

The evaluation of the effects of flooding at the landfill suggests that very little inundation of actual waste-filled areas occurs at less than the 50-year flood level (designated at 444 feet amsl). The 100-year flood level (designated at 447 feet amsl) would cover approximately 25 to 50 percent of the landfill and the 500-year flood level (designated at 452 feet amsl) would essentially cover the entire site.

Under the 100-year flood conditions, the northern tract, small portions of the central tract and approximately one-half of the southern tract where the large depression currently exists would be affected. Some scouring of the landfill cover would be expected, as well as increased infiltration resulting in increased leachate production within the landfill. Groundwater reversal could carry landfill contaminants into Riverside Gardens if the period of flooding were sufficiently long.

The western boundary of the Landfill abuts the Ohio River, with a portion of this boundary being characterized by a moderate to steep slope. This close proximity to the river, in conjunction with the steepness of slope, gives this particular area of the site a potential for erosion and bank failure during flood stages on the Ohio River that occur as frequently as every 1.2 years. Visual inspection of the bank



ONSITE WETLANDS AND STANDING WATER
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

LEGEND

- OPEN POND
- MARSHY AREA
- STANDING WATER
- AREA POTENTIALLY DRAINING TO POND

FIGURE 6

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reveals exposure of vegetation root systems, making the erosion due to river flow readily apparent.

Water quality analyses were performed on surface water samples collected from a marsh and the pond in the southern tract and small areas of standing water in the northern and central tracts. The evaluation of the results suggested that very low levels of contamination were present. A comparison of water levels in the pond in the southern tract with nearby groundwater levels did not suggest the potential for groundwater discharge to the pond. Comparison of nearby groundwater quality to surface water quality in the pond confirmed the above evaluation based on water levels. In addition, the levels of contaminants in the pond did not suggest that leachate was being discharged to the pond in significant quantities.

2.3 Sediments and Surface Soils

The sources of contamination of surface soils include past activities such as dumping or staging of drums during removal operations and discharge of leachate seeps. The surface soils are not considered to be a significant source of contamination since the site has been closed and covered.

The migration routes are expected to be the same as those for surface water. Runoff containing eroded surface soils will be discharged to the Ohio River or the pond in the southern tract. Since the level of contamination of the cover materials is expected to be uniform throughout the site, except as a result of specific past activities, the sediments in the pond in the southern tract are expected to be characteristic of transported surface soils.

The potential "hot spot" surface soils associated with indiscriminate dumping were evaluated through the collection of soil samples in areas exhibiting visual evidence of vegetative stress. It is assumed that the majority of the landfill cover is less contaminated than these "hot spot" soils. Potential public contact with these soils is not expected since the areas were identified based on visual evidence and such soils would probably be avoided at the site.

Soil and sediment analyses were performed as part of the RI. The evaluation of the results of these analyses suggested little variation in the concentration of contaminants in the surface soils and sediment from those found in offsite soils in Riverside Gardens. This similarity confirms that the landfill was probably covered with local soils.

2.4 Air and Toxic Gases

The potential source of contaminants to ambient air is the release of toxic organic compounds produced by the decomposition of landfill wastes. The subsurface migration of landfill gases into Riverside Gardens was documented from 1975 to 1979. The production of these gases can be expected to decrease with time as the landfill stabilizes, but studies conducted in 1984 confirm that the gases are still being produced. Air investigations are currently being conducted by EPA both on and off the site to investigate any potential air problems.

3.0 EXPOSURE PATHWAYS

Exposure pathways are the routes by which suseptible receptors may be exposed to a contaminant. Primary exposure routes include ingestion, inhalation, and dermal contact. Ingestion may take the form of direct exposure through drinking or eating materials which are contaminated or may involve indirect routes such as use of contaminated water for food preparation. Direct inhalation exposure results from breathing air which has become contaminated through volatilization, release of gas-phased contaminants, or entrainment of airborne particulates. In the case of particulate inhalation, the physical size of the particulate as well as chemical characteristics play a major role in determining the severity of the exposure since the size range for "respirable" particulates is very restricted. Dermal exposure may result from direct contact with contaminated water, soil or other material, or may involve indirect contact such as transfer of contaminants from original sources to clothing and furniture and subsequent skin contact. Any of these exposure routes may result in an acute exposure, which involves short time duration and frequency of exposure or chronic exposure, which is of longer duration

and is continuous or frequent. The major exposure pathways of concern to receptors at the Lees Lane Landfill Site are shown for each media in Table 6.

3.1 Migration Routes

A wide array of contaminants were found at low levels in the various media at the Lees Lane Landfill Site. Most were found only sporadically and were not considered representative of the typical site conditions. Some of these contaminants included plasticizers (phthalates), heavy metals, pesticides, and solvents. The site contaminants could potentially reach receptors through inhalation, ingestion or contact with contaminated media. The various transport routes which could potentially deliver the contaminants to the receptors include subsurface gas migration, particulate or gas migration in open air, surface water runoff, and groundwater discharge. Receptors could also be affected by direct dermal contact with locally contaminated surface soils.

3.1.1 Groundwater

Pollutant movement in the groundwater system is the major transport route to potential offsite receptors and will be examined more closely in this Assessment. A small number of shallow, private drinking water wells are located in the Riverside Garden subdivision, east of the site. No elevated contaminant levels were found in these wells. Two deep industrial process wells are also located north and south of the site and are operated by Borden and Louisville Gas and Electric. Analyses conducted during the RI did not reveal any elevated levels of hazardous contaminants in the wells. Two public water supply wells withdrawing from the deeper portions of the aquifer are located on the Indiana side of the Ohio River. No contaminants typical of the site were found at elevated levels in these wells, although manganese was observed in excess of the MCL defined by the Secondary Drinking Water Regulations. Manganese, iron and chromium appear to be widespread in the deeper portions of the aquifer. These substances were observed in upgradient monitor wells, onsite monitor wells, and the Indiana public water

TABLE 6
EXPOSURE PATHWAYS
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

Affected Area	Release Mechanism	Affected Media	Migration Route	Exposure Route	Receptors
Offsite	Leachate	Groundwater	Into Ohio River	Biological Uptake	Downstream Users
			Under Ohio River	Ingestion	Ind. PWS Users
			Into Riverside Gardens	Ingestion	Residental Well Users
	Runoff	Surface Water	Into Ohio River	Biological Uptake	Flora and Fauna
		Sediments	Into Ohio River	Biological Uptake	Flora and Fauna
		Surface Soil	Into Ohio River	Biological Uptake	Flora and Fauna
	Gas Production	Gas Migration	Into Riverside Gardens	Inhalation	Homes in Riverside Gardens
		Air	Into Riverside Gardens	Inhalation	Residents in Riverside Gardens
Onsite	Unrestricted Access	Surface Water	To Pond in S. Tract	Direct Contact	Recreational Users
		Sediments/Soils	To Pond in S. Tract	Direct Contact	Recreational Users
		Air	Thru Cover Material	Inhalation	Recreational Users

supply wells. Although the site may contribute to the elevated levels, it does not appear to be the sole source.

3.1.2 Surface Water

The contaminants in the onsite surface soils could potentially reach offsite receptors by migrating with surface water runoff. In the northern and central tracts, surface water is primarily discharged to the groundwater due to topography and permeable cover soils. Surface water runoff in the southern tract, however, flows into the low-lying pond water area. Based on topographic features, the pond area appears to be a sink for some of the pollutants in the surface water runoff. Even so, pollutant levels in standing water and in sediments in these areas are relatively low or nonexistent. Surface water runoff from the site is to the Ohio River.

3.1.3 Sediments and Surface Soils

In general, the sediments and surface soils at the site are not contaminated. Low levels of two contaminants chromium, and lead were observed in two localized areas. These areas were along the access road in the central tract, possibly indicating sporadic dumping. Receptor contact with these areas could potentially lead to exposure, however, the "hot spots" do not represent a serious problem since they are not typical of general site conditions. They can be easily covered to minimize receptor exposure.

3.1.4 Subsurface Gas Migration

In early 1975, a gas was detected in homes and septic tank vents in the Riverside Gardens residential subdivision. Analyses conducted by the EPA and SCS Engineers between 1975 and 1978 revealed the gas was composed primarily of carbon dioxide and methane. In areas close to the Lees Lane Landfill Site, methane concentrations were commonly above 20 percent volume in air, which is sufficient to cause an explosion hazard. Analyses for gases other than methane were also

conducted and revealed the presence of a number of toxic gases in test well headspaces throughout the residential area. Some of the gases observed included vinyl chloride, benzene, dichloroethane, ethylbenzene, and toluene. In 1980, a gas collection system was installed at the site boundary adjacent to the residential area to intercept gases migrating in the subsurface. Testing in 1984 by IT Corporation indicates that the gas collection system appears to have temporarily eliminated the gas problem to the nearby residences. Subsurface gas migration is not expected to affect residences as long as the system is operating properly.

3.1.5 Ambient Air

No representative sampling of the air medium was undertaken during the RI. However, residents of the Riverside Gardens subdivision have voiced significant concerns about adverse health problems. They feel that these problems may be related to chronic exposure to gases being emitted from the landfill followed by subsurface migration into their homes or air transport to the ambient air of the subdivision. EPA is currently conducting an air sampling program both on and off the site. Results of this investigaiton will be presented in a separate report.

3.2 Receptors

The Lees Lane Landfill Site is located in a mixed land use area. Industrial operations are located immediately north and south of the site, while a large residential area is located immediately to the east. An estimated 1,470 people live within a one-mile radius of the site (NUS, 1983). A large tract of undisturbed land in the Mill Creek Cutoff area and the diversity of habitats on the Lees Lane Landfill Site suggest that numerous environmental receptors could also be affected by contaminants at the site.

3.2.1 Groundwater

Most residents in the area use public water, however, approximately eleven homes still use domestic wells tapping the alluvial aquifer. Of these eleven wells, only

eight are used for drinking water. Industrial wells north and south of the site and Indiana public water supply wells on the west side of the Ohio River withdraw water from the alluvial aquifer. The public water supply wells in Indiana supply the Edwardsville Water Company which serves approximately 1700 connections directly and supplies water to two other water companies. Ingestion of contaminated groundwater is the primary exposure route to the human receptors near the landfill site.

3.2.2 Surface Water

The most likely receptors for onsite surface water contaminants would include people using the site for recreation and biota living or feeding in the wetland/open water areas or in the nearshore area of the Ohio River. The dominant exposure routes would be through dermal contact to humans and ingestion to biota.

3.2.3 Sediment and Surface Soils

Receptors most likely to be exposed to sediment and surface soil contaminants include trespassers, hunters, children, remediation workers, and biota at the site. Short-term dermal contact with contaminated soils and inhalation of contaminated dust are the most probable exposure routes.

3.2.4 Air

Receptors of chronic levels of ambient air contaminants include those people living or working near the site. Hunters and other site visitors could be subject to acute levels of exposure. Based on distances from the site, potential receptors of adverse affects of subsurface migration of contaminants is expected to be limited to residents of Riverside Gardens. The exposure route is inhalation of hazardous gases or contaminant-laden particulates.

3.2.5 Biota

Biota residing at or migrating through the site may become exposed to contaminants through all three exposure routes. The woodland/brushland ecotones should contain an abundant rodent population, based on nearby ecological surveys (COE, 1982), which could also attract predators to the site. Although no large waterfowl or wading bird populations frequent the area, the marsh and open water areas could potentially attract wildlife. The primary human receptors that could be potentially affected by contaminated biota are hunters and fishermen who may visit the site.

3.3 Potential Health Effects

The evaluation of the potential health effects of the critical contaminants identified at the Lees Lane Landfill Site includes an examination of the environmental fate and the chemical and biological toxicological properties of each contaminant, a review of compound-specific environmental criteria and a summary of potential toxic effects to the general population near the site.

Chemical toxicity results from acute and chronic exposure to the contaminants. Acute exposure is a single short-duration exposure and chronic exposure involves repeated or continuous exposure to low levels of the contaminant. Established environmental health criteria for the critical contaminants are provided in Table 7. The biological toxicity effects examined for the critical contaminants include carcinogenicity, mutagenicity and teratogenicity. The carcinogenicity of a chemical is based on its potential to induce cellular changes resulting in cancer formation. Mutagenicity is the ability of chemicals to cause changes in genetic materials in ways that can be transmitted during cell division, while teratogenicity is based on a chemical's ability to cause birth defects. Table 8 provides the biological toxicity characteristics of the contaminants of concern. Additional information on the chemical and biological toxicity of the critical contaminants is provided in the Appendices.

These classifications are generally qualitative but represent the best information available at this time. Care has been taken in interpretation of results,

TABLE 7
CRITERIA FOR CRITICAL CONTAMINANTS
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

Compound	Human Health Criteria (Ingestion)	Threshold Limit Values Time Weighted Average (TLV-TWA) (Inhalation) (a)	Aquatic Life Criteria
Lead	50.0 ug/l (b)(c)	0.15 mg/m^3	Depends on water hardness. 170 ug/l at hardness measured as 100 mg/l calcium carbonate.
Arsenic	50.0 ug/l (b)(c) 22,000 ug/l (d)	0.2 mg/m^3	440 ug/l
Benzene	6.6 ug/l (d)	30 mg/m^3; 10 ppm	-
Chromium	50.0 ug/l (b)(c)	0.05 mg/m^3	21 ug/l

(a) TLVs for Chemical Substances in the Work Environment Adopted by ACGIH 1984-1985.
(b) National Interim Primary Drinking Water Standard Maximum Contaminant Level
(c) Kentucky Water Quality Standard
(d) Criterion associated with a human lifetime cancer risk of 10^{-5}

\- Not established.

Source: See Appendices.

TABLE 8
BIOLOGICAL TOXICITY FOR CRITICAL CONTAMINANTS
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

Contaminant	Maximum Observed Concentration* (ug/l)	Carcinogenicity	Mutagenicity	Reproductive/Teratogenic Effects
Lead CAS No. 7439-92-1	150	Certain lead compounds are carcinogenic in test animals. IARC considers the animal evidence of dubious significance to man.	No excess chromosome damage in cultured leukocytes obtained from cows accidentally poisoned with lead.	No evidence that lead has a teratogenic effect in man. Has been shown to have teratogenic effects in test animals.
Arsenic CAS No. 7440-38-2	87	Ingestion and inhalation exposures increase the risk of skin and lung cancer in humans.	Chromosomal aberrations noted in human cell cultures exposed to sodium arsenate.	Sodium arsenate induces developmental malformations in a variety of test animals.
Benzene CAS No. 71-43-2	450	Carcinogenic in mice and rats. Suspected of causing leukemia in humans.	Causes microlesions in Salmonella typhimurium, Drosophila melanogaster. Causes macrolesions in rats and man.	Causes cleft palate, micrognathia and agnathia in mice.
Chromium CAS No. 7440-47-3	640	Suspected of causing lung cancer in humans. Rats injected with calcium chromate developed tumors at the point of injection.	Causes microlesions in E. coli. Causes chromosomal aberrations in mouse fetal cells.	Insufficient data available to make an evaluation of teratogenicity.

* Concentrations shown are for groundwater.

Source: See Appendices.

extrapolation of animal toxicity test data to human application, and development of qualitative conclusions based on incomplete and inconclusive data.

The qualitative risk assessment of environmental and health impacts which follows combines currently available health effects data with an evaluation of a site-specific determination of exposure probability.

3.3.1 Lead

Lead is a soft, gray, heavy, ductile metal which is found in nature in the sulfide (galena), the sulfate (anglesite) and the carbonate (cerrusite) mineral deposits. Approximately one-half of the lead produced is used in storage batteries, one fifth in gasoline additives (decreasing, however), and the remainder in lead-containing alloys, solders, pigments and ceramics.

Environmental Fate

Lead is considered insoluble in water, but may be solubilized in some acids. The solubility of lead compounds in water increases as the pH and the concentration of dissolved salts decreases. Hem and Durum (1973) found the solubility of lead to range from 10,000,000 ug/l at pH 5.5 to 1 ug/l at pH 9.0. Lead does not move readily through normal groundwater or surface water because it forms insoluble carbonates and sulfates and binds to organic ligands of flora and fauna.

Food has traditionally been considered the major source for lead exposure in humans. Surface deposition of lead on plants and vegetables and uptake via roots are the prime pathways. There is little evidence for the biomagnification of lead in the food chain; thus, fish are not usually considered a significant source.

Hazard Assessment

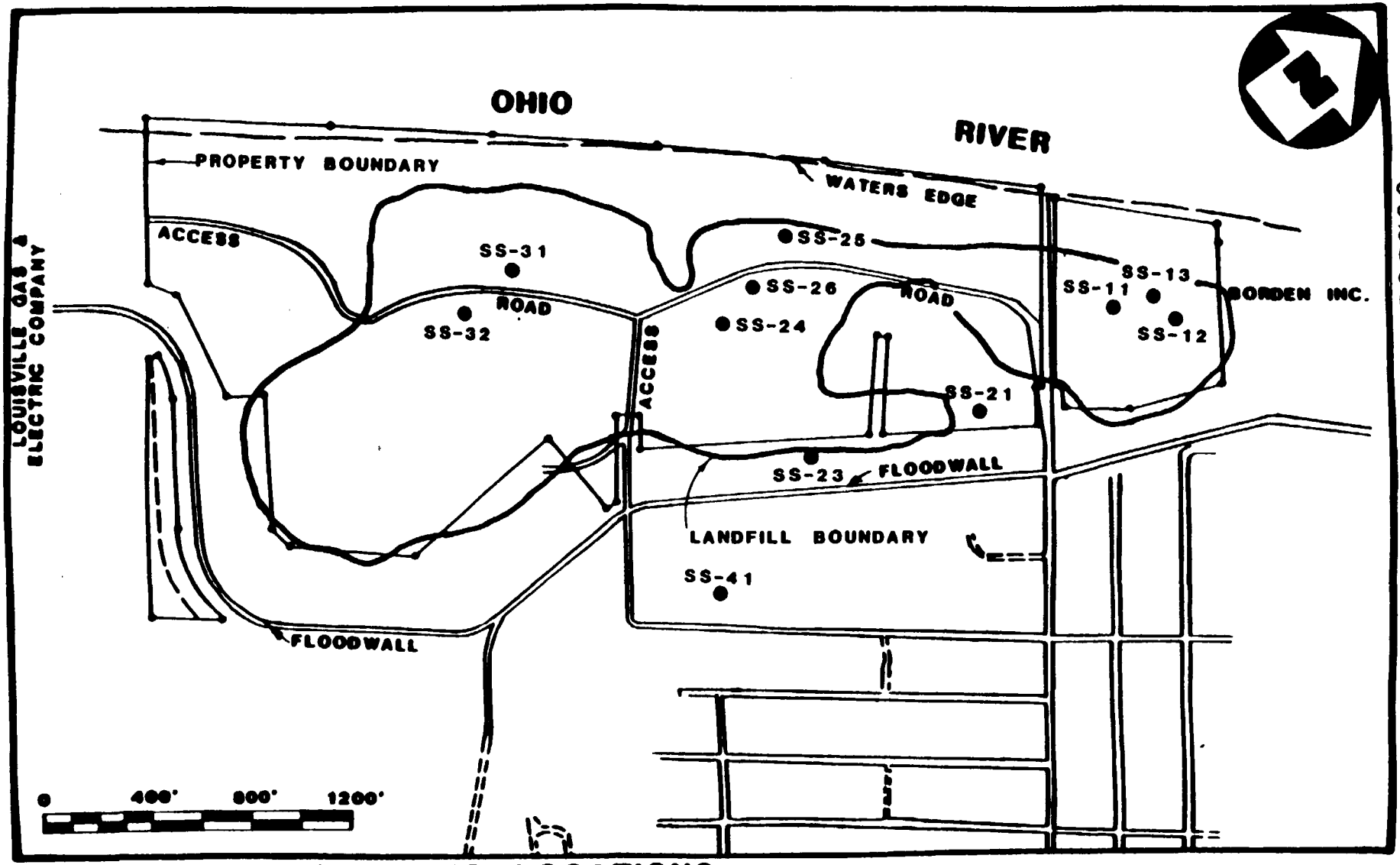
Lead is commonly found in nature, being a natural component of the earth's crust. Most natural groundwater has concentrations ranging from 1 to 10 ug/l. In a survey

of 969 U.S. water systems, 1.4 percent were found to have concentrations of lead in excess of the 50 ug/l drinking water maximum contaminant level (McCabe, 1970). Potentially hazardous lead exposures to man via drinking water have usually been linked to lead-lined storage tanks or pipes.

During the RI, lead was a common constituent at the site. In a sample of surface waste (SS-22) from the central tract, the estimated lead concentration was 20 mg/kg. In background soils (SS-41), the estimated lead concentration was 50 mg/kg. Estimated lead levels in "hot spot" soil samples ranged from 50 to 2,000 mg/kg. The highest concentration was found at SS-23 in the central tract. No criteria have been establised for lead in soils, however the Department of Health and Human Services (DHHS, 1983) indicate that naturally occurring lead ranges from 2 to 200 mg/kg in soils nationwide. The surface soil sampling locations at the Lees Lane Landfill Site are shown in Figure 7.

Lead concentrations in surface water on the site ranged from undetected to an estimated concentration of 10 ug/l, while the concentrations ranged from 10 to an estimated 100 mg/kg in bottom sediments. No lead was detected in nearshore Ohio River surface water samples. The ambient water quality criterion to protect freshwater aquatic life is dependent on water hardness and ranges from 74 to 400 ug/l total recoverable lead. Lead concentrations are well with in this criterion and the 500 mg/kg alert level established by the USGS (1977) for river bottom materials. The surface water and sediment sampling locations and the Ohio River and well point sampling locations are shown in Figures 8 and 9, respectively.

The lead concentration in the shallow background well (MW-01) was 7.2 ug/l. The lead concentration in onsite shallow monitoring wells ranged from undetectable to 150 ug/l. The latter concentration was detected in LL-9, which also had elevated chromium and arsenic levels. The lead level in the deep upgradient monitoring well (MW-02) ranged from 28 to an estimated 23 ug/l between December 1984 and January 1985. The lead concentrations in the deep onsite monitoring wells ranged from 11 to an estimated 68 ug/l. The latter concentration was detected in MW-03. No lead was observed in the industrial wells adjacent to the site, however the



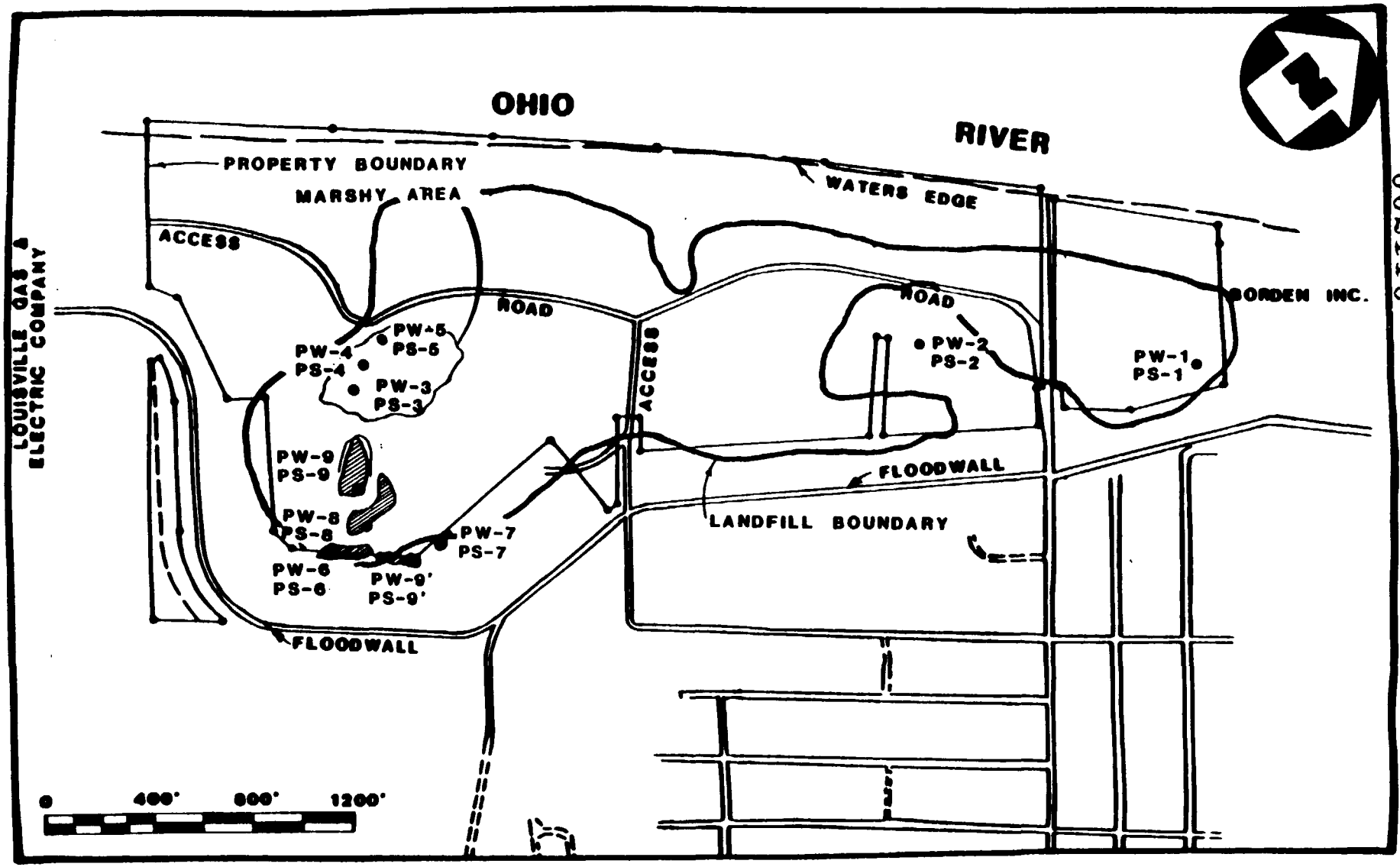
SURFACE SOIL SAMPLING LOCATIONS
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

LEGEND

● - SAMPLE LOCATIONS

FIGURE 7

NUS CORPORATION
A Halliburton Company



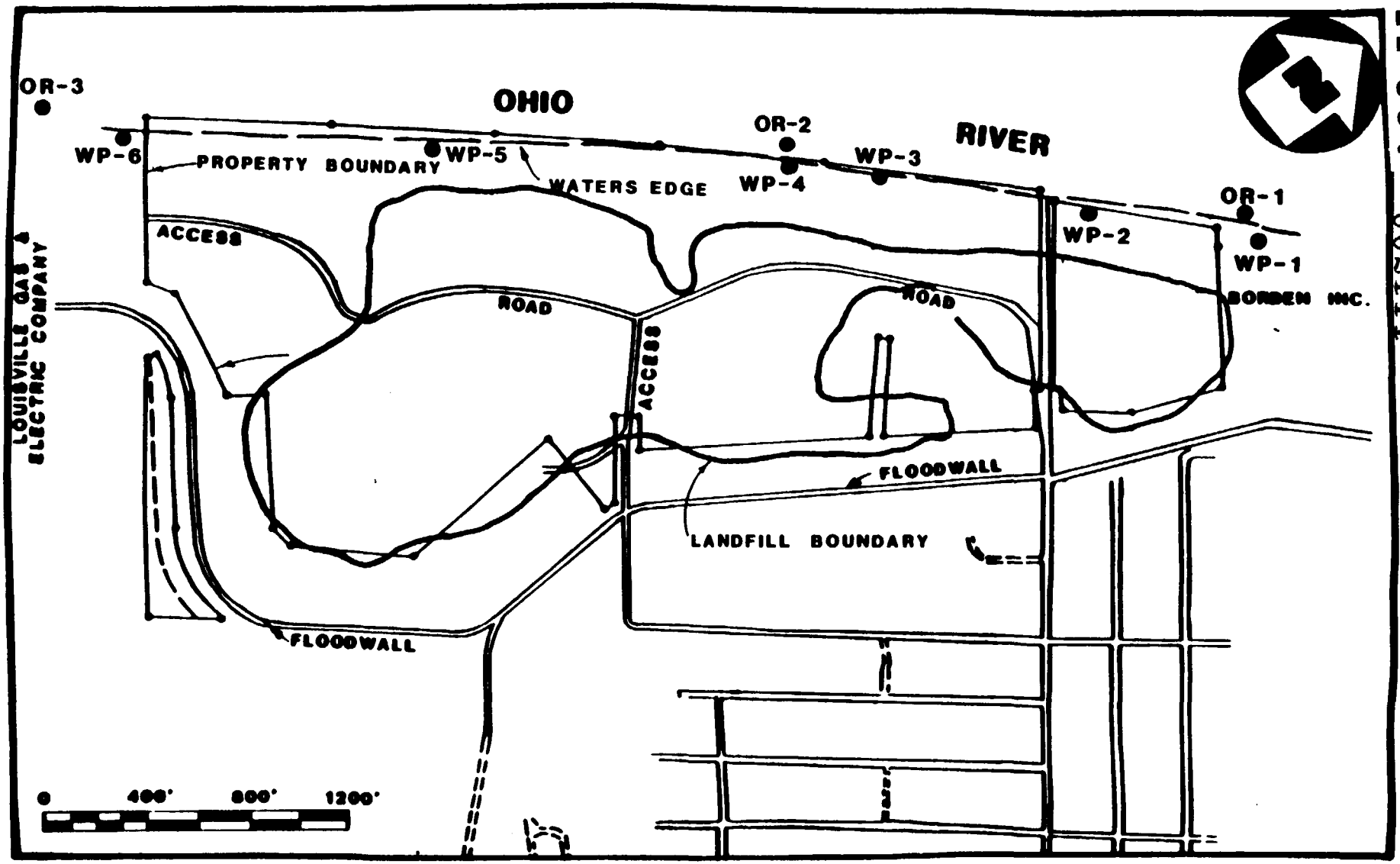
ONSITE SURFACE WATER AND SEDIMENT SAMPLING LOCATIONS LEES LANE LANDFILL SITE JEFFERSON COUNTY, KENTUCKY

LEGEND

- OPEN PONDED WATER
- SAMPLE LOCATION

FIGURE 8

NUS CORPORATION
A Halliburton Company



OHIO RIVER AND WELL POINT SAMPLING LOCATIONS
LEES LANE LANDFILL SITE
JEFFERSON COUNTY , KENTUCKY

LEGEND
● SAMPLE LOCATIONS

FIGURE 9

NUS CORPORATION
A Halliburton Company

concentrations in the Indiana public water supply wells ranged from undetectable to 10 ug/l. The lead concentrations in the private residential wells ranged from undetectable to 32 ug/l. In only two instances were the lead concentrations in excess of the National Interim Primary Drinking Water Regulation (NIPDWR) maximum contaminant level of 50 ug/l (EPA, 1977.). These levels were 150 ug/l in LL-9 and an estimated 68 ug/l in MW-03. The well sampling locations at and near the site are shown in Figure 10.

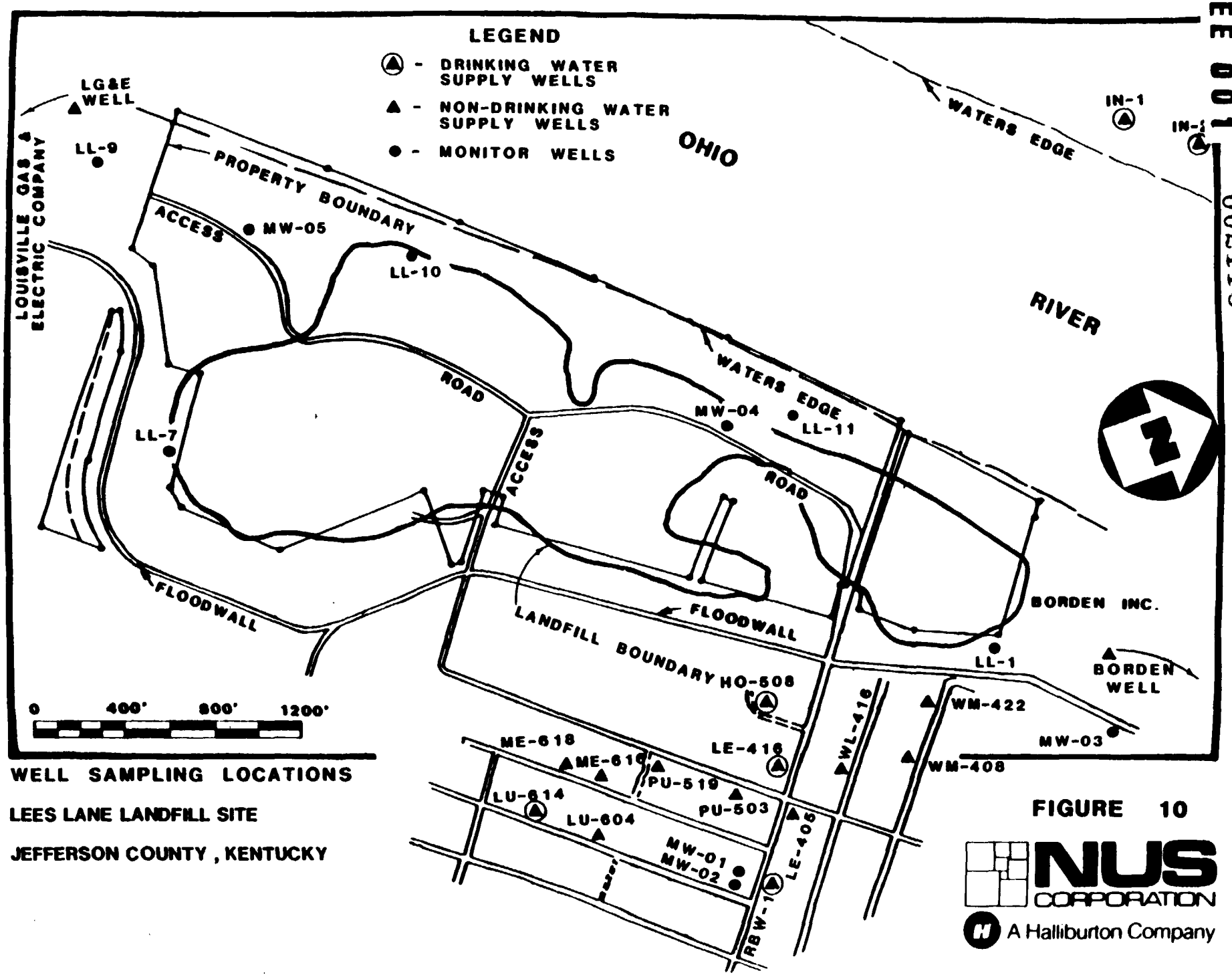
In general, lead was detected at low levels in the soil and water media at the Lees Lane Landfill Site. Based on the observed levels, it is unlikely that lead represents a significant public health threat.

3.3.2 Arsenic

Arsenic is a silver-gray, brittle, crystalline, metallic-looking substance. It exists in three allotropic forms, the yellow (alpha), black (beta), and the grey (gamma) modifications. It is insoluble in water but is soluble in nitric acid. Arsenic is used as an additive for metals, especially lead and copper (90 percent); in electronic devices (7 percent) and as a chemical intermediate for arsenicals used in veterinary medicines (3 percent).

Environmental Fate

Elemental arsenic is seldom encountered in natural waters and is considered of low toxicity because of its virtual insolubility in water or body fluids (EPA, 1976). Soluble inorganic arsenate (pentavalent) compounds predominate under normal conditions over the less stable arsenite (trivalent) compounds. Waters of low pH and low dissolved oxygen favor the formation of lower oxidation state compounds such as arsenite. More basic, less oxygenated waters favor the formation of pentavalent arsenates (Ferguson and Gavis, 1972).



WELL SAMPLING LOCATIONS

LEES LANE LANDFILL SITE

JEFFERSON COUNTY , KENTUCKY

FIGURE 10

NUS CORPORATION

A Halliburton Company

Arsenic is present in nearly all foods, with fish and seafood containing the most and fruits the least. It is estimated that the intake rate for arsenic from the consumption of terrestrial foods is 1 mg/yr.

Hazard Assessment

Arsenic is a naturally occurring element which has been detected in surface water, groundwater, and soils. In a survey of 1577 surface waters, 87 contained arsenic in concentrations ranging from 5 to 336 ug/l with a mean of 64 ug/l (Kopp, 1969). According to the National Academy of Science (1977), most of the high levels of arsenic in surface waters are attributable to industrial contamination, with smelters and power plants also important sources. Based on toxicological studies, EPA has established an ambient water quality criterion of 440 ug/l of total recoverable trivalent arsenic for the protection of aquatic life.

During the RI, arsenic was observed at low levels in the soil and water media. The arsenic concentration in a sample of waste material (SS-22) was 190 ug/l, while it was 24 mg/kg in background soils (SS-41). The arsenic concentration in surface soils ranged from undetected to 25 mg/kg. The DHHS (1983) indicates arsenic concentration in surface soils generally ranges from 0.1 to 40 mg/kg.

No arsenic was reported for onsite surface waters, however arsenic concentrations ranged from 5.4 to 27 mg/kg in bottom sediments. These concentrations are well below the 200 mg/kg alert levels established by the USGS (1977) for river bottom material.

Arsenic was not detected in the shallow upgradient monitoring well (MW-01). The arsenic concentration in onsite shallow monitoring wells ranged from undetectable to 87 ug/l. The latter concentration was detected in LL-9, which also had elevated chromium and lead levels. Arsenic was also not detected in the deep upgradient monitoring well (MW-02), and concentrations in the lower portion of the groundwater onsite ranged from undetected to 8.1 ug/l. No arsenic was detected in private residential wells and arsenic was not reported for the industrial process

wells or the Indiana public water supply wells. In only one instance was the arsenic concentration in groundwater in excess of the NIPDWR maximum contaminant limit of 50 ug/l (EPA, 1977). This was at monitoring well LL-9 which had an arsenic concentration of 87 ug/l.

In summary, arsenic was detected at low levels in the soil and water media at the Lees Lane Landfill Site. Based on the levels observed, it is unlikely that arsenic represents a significant public health threat.

3.3.3 Benzene

Benzene is a liquid hydrocarbon produced principally from the distillation of petroleum by catalytic reforming of light naphthane and isolation by distillation or solvent extraction. Benzene is used extensively in the chemical industry as a solvent for industrial extraction and rectification. Other uses for benzene include: degreasing and cleaning; as a solvent in the rubber industry; as an anti-knock fuel additive; and in the manufacture of styrene, cyclohexane, detergents, and pesticides.

Environmental Fate

Benzene (C_6H_6) is a volatile, colorless liquid with a molecular weight of 78.1. It is a highly flammable liquid with a characterisitic odor. It has a boiling point of 80.1°C and a flash point of -11°. Benzene is slightly soluble in water (0.178 gms/100 ml at 20°C) and has a density of 0.879. It also has vapor density of 2.77.

Benzene is photo-oxidized in the air and undergoes rapid bacterial degradation in the soil.

Hazard Assessment

Benzene was selected as a critical contaminant since it is a human carcinogen and was observed in a variety of media at and near the site. No benzene was found in background or onsite surface soils.

Benzene was observed in only one surface water/sediment sample at the site. It was detected at 5J ug/l in standing water in the central tract (PW-2) and at 15J mg/kg in bottom sediments at the same location (PS-2). No criteria are available for surface water, although acute toxicity in freshwater aquatic life can occur as low as 5,300 ug/l and chronic effects can occur at even lower concentrations (EPA 1980). For humans, the ambient water concentration should be zero due to its carcinogenic potential. Since these levels may not be attainable, according to EPA (1980), the levels which may result in incremental increase of cancer risk over the lifetime are estimated at 10^{-5} , 10^{-6}, 10^{-7}. The corresponding recommended criteria are 6.6 ug/l, 0.66 ug/l and 0.066 ug/l, respectively. If the above estimates are made for consumption of aquatic organisms only, excluding water consumption, the levels are 400 ug/l, 40.0 ug/l, and 4.0 ug/l, respectively (EPA, 1980).

Benzene was observed only in two groundwater samples during the RI. A benzene concentration of 450 ug/l was observed in the shallow groundwater which discharges into the Ohio River (WP-3). A benzene concentration of 20 ug/l was also observed in the deep upgradient monitoring well (MW-02). Using the one-hit linear model, the excess cancer risk for people exposed to 20 ug/l of benzene over a lifetime is 3×10^{-5}. This indicates a probability of 3 additional cases of cancer for every 100,000 people exposed. Although the benzene observations are a matter of concern, they occur sporadically and do not appear to be representative of general site conditions.

3.3.4 Chromium

Chromium is a metallic element which exists in several valance states. Chromium is used in the manufacturing of chrome-steel, chrome-nickel-steel alloys, cast iron,

super alloys, and other alloys. Chromium salts are used extensively in the metal finishing industry and electroplating; they are also used as cleaning agents, and as mordants in the textile industry (Sittig, 1985).

Environmental Fate

The hexavalent and trivalent chromium compounds are the biologically and environmentally significant forms of the metal, but they have different chemical characterisitics. Hexavalent chromium is very soluble in natural waters. It is also a strong oxidizing agent in acidic solution, although it is relatively stable in most natural waters.

Trivalent chromium tends to form stable complexes with negatively charged organic or inorganic chemicals and thus its solubility and toxicity vary with water quality characteristics such as hardness and alkalinity.

Hazard Assessment

Chromium was widespread and was found in elevated levels in the soil and water media at the Lees Lane Landfill Site. In a sample of surface waste at the site (SS-22), the estimated chromium concentration was 5,000 ug/l, while in background soils (SS-41) the estimated concentration was 20 ug/l. Estimated chromium concentrations ranged from 10 to 2,000 ug/l in "hot spot" soil samples at the site. The highest chromium concentrations were detected in the soils in the central tract. Station SS-23 contained an estimated chromium level of 400, while stations SS-24 and SS-26 contained 2,000 and 900, respectively. The DHHS (1983) indicates the range of chromium normally found in surface soils is 5 to 3,000 mg/kg. The chromium levels detected at the Lees Lane Landfill are well within this range.

Chromium levels ranged from undetectable to 6.2 ug/l in surface waters at the site and no chromium was detected in the nearshore area in the Ohio River. Bottom sediment chromium levels ranged from 9.8 to 30 mg/kg. In order to protect freshwater aquatic life, a concentration of 21 ug/l hexavalent chromium should not

be exceeded according to EPA (1980). The USGS alert level (1977) for chromium in river bottom material is 200 mg/kg. Neither of these criteria were exceeded.

Chromium was a common constituent in the groundwater at and near the site. The chromium level in the shallow upgradient well (MW-01) was 43 ug/l. The concentrations ranged from 12 to 140 ug/l in the shallow groundwater at the site. The 140 ug/l level was observed in well LL-9, which also contained elevated levels of arsenic and lead. Additionally, 900 ug/l chromium was observed in LL-9 in April 1981. The deep upgradient monitoring well (MW-02) had a chromium concentration range of 120 to an estimated 57 ug/l between December 1984 and January 1985. In the deep monitoring wells, the chromium concentration ranged from an estimated 30 to 640 ug/l, with a mean concentration of 312 ug/l. Chromium was not observed in the Borden or L G & E industrial wells, however, a concentration of 12 ug/l was noted in an Indiana public water supply well (IN-1). Chromium was not detected in private wells sampled in the Riverside Gardens area. Although not directly applicable, the NIPDWR Maximum Contaminant Level for chromium is 50 ug/l. It is apparent that the deep upgradient well (MW-02) and many of the onsite wells had concentrations greatly in excess of this standard. It does not appear that the site is the only source of the chromium since it was found in elevated levels in the upgradient wells (MW-01 and MW-02). The site, however, does appear to be contributing to the problem.

Since MW-03 contained the highest chromium concentration (640 ug/l), it will be used to evaluate potential adverse health effects. The acceptable daily intake (ADI) of chromium for man has been calculated to be 83 ug/l. This compares to a daily intake of chromium through drinking water of 1,280 ug/day/person for MW-03. Although unlikely, it is possible that drinking water containing 640 ug/l over a period of several years may lead to an increase in the chromium concentrations of the liver and spleen. Chronic toxicological effects are possible at this level based on animal studies. No dermal effects from bathing in water containing 640 ug/l are anticipated, although chromium is recognized as a potent sensitizer of skin.

4.0 ENVIRONMENTAL EFFECTS

The diversity of habitats at the Lees Lane Landfill indicate the site could contain an abundant faunal population. Small mammals such as field mice and rabbits residing in the grass areas would be the most predominate species. Deer and other larger mammals may also frequent the riparian woods and woodland/brushland ecotone. Although large populations of waterfowl or wading birds are not known to inhabit the area, the marsh/open water areas could potentially attract them. The environmental receptors can be exposed to site contaminants by all exposure routes. The most important route, however, is ingestion.

Table 9 provides the lethal concentrations or doses for various organisms ranging from sensitive aquatic invertebrates to small mammals. It is apparent from the table that environmental factors, such as water hardness, play an important role in determining the aquatic toxicity of the heavy metals. Heavy metals are much more toxic to aquatic life in soft water than in hard water. For example, in hard water the LC_{50} for lead in sunfish is 442,000 ug/l, while in soft water the LC_{50} is only 23,800 ug/l. This is also apparent with chromium.

None of the critical contaminants are readily bioaccumulated in the food chain, although chromium, lead, and arsenic can accumulate in the tissues of exposed organisms. Many studies suggest that metals biomagnify similar to DDT. Connell and Miller (1985) report that food chain enrichment of metals does not occur, except for mercury. Rather, organisms exposed to the highest metal concentrations generally contain the greatest amounts. For example, detritus-feeders exposed to contaminated bottom sediments can contain higher metal amounts than upper trophic level feeders. Other factors determining metal concentrations in biota include age and preferential uptake and elimination of different metals by the body.

Levels of the critical contaminants in surface waters and groundwater were well below the lethal concentrations shown in Table 9, and do not represent a significant threat to environmental receptors at these concentrations.

TABLE 9
BIOTA ACUTE TOXICITY FOR CRITICAL CONTAMINANTS
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

Critical Contaminant	Invertebrate	Coarse Fish	Game Fish	Small Mammals
Lead	612 ug/l = LC_{50} (soft water) 1,910 ug/l = LC_{50} (hard water) (Daphnia magna)	31,500 ug/l = LC_{50} (soft water) N/A (hard water) (Goldfish)	23,800 ug/l = LC_{50} (soft water) 442,000 ug/l = LC_{50} (hard water) (Bluegill)	520 mg/kg oral TDLo (Rat)
Arsenic	5,278 ug/l = LC_{50} (Daphnia magna)	26,042 ug/l = LC_{50} (Juvenile Goldfish)	41,760 ug/l = LC_{50} (Bluegill)	
Benzene	380,000 ug/l = LC_{50} (Daphnia magna)	34,000 ug/l = LC_{50} (Goldfish)	22,000 ug/l = LC_{50} (Bluegill)	5,600-5,700 mg/kg = LD_{50} (Rat)
Chromium (CR III)	2,000 ug/l = LC_{50} (soft water) 58,700 ug/l = LC_{50} (hard water) (Daphnia magna)	4,100 ug/l = LC_{50} (soft water) N/A (hard water) (Goldfish)	7,460 ug/l = LC_{50} (soft water) 71,900 ug/l = LC_{50} (hard water) (Bluegill)	1,870 mg/kg = oral LD_{50} (Rat)
(CR VI)	17,300 ug/l = LC_{50} (soft water) 40,600 ug/l = LC_{50} (hard water) (Snail)	37,500 ug/l = LC_{50} (soft water) 124,000 ug/l = LC_{50} (hard water) (Goldfish)	118,000 ug/l = LC_{50} (soft water) 133,000 ug/l = LC_{50} (hard water) (Bluegill)	110 ug/m^3 Inhalation TClo (Hamster)

TCLo - Toxic Concentration Low - the lowest concentration of a substance that has produced any toxic effect.
LC_{50} - Lethal Concentration Fifty - concentration of a substance, exposure to which is expected to cause the death of 50% of test animals.
LD_{50} - Lethal Dose Fifty - concentration of a substance, exposure via any route but inhalation is expected to cause the death of 50% of test animals.
DLo - Lethal Dose Low - the lowest dose of a su tance, introduced via any route but inhalation, that has been reported to cause death in humans or animals.
N/A - Not Available

Sources: Verschueren, K. 1983.
Sax, N.J. 1984.

In summary, the concentrations of the critical contaminants observed during the remedial investigation do not represent a significant threat to the environmental receptors at the Lees Lane Landfill Site. Biota in continued direct contact with elevated contaminant levels in selected "hot spot" soil areas may experience symptoms of chronic toxicity, however, no acute toxicological effects would be expected at the current contaminant levels.

5.0 SUMMARY AND CONCLUSIONS

The most significant potential public health problem identified by the Endangerment Assessment at the Lees Lane Landfill Site is related to the elevated chromium levels detected in the groundwater. Although the site is contributing to the elevated levels, it is not the only source since upgradient wells also contained elevated levels. Chromium was not detected in residential wells east of the site. Since groundwater flow is predominantly toward the Ohio River it is unlikely the residential wells will be affected in the future. Chromium was not detected in the industrial process wells north and south of the site, however it was found at low levels in the Indiana public water supply wells across the Ohio River. It is not known if this chromium is related to elevated levels at the landfill.

Due to numerous complaints from Riverside Gardens residents regarding odors in the air, EPA is conducting an on-going air investigation at and in, the vicinity of the site. After sufficient data have been collected and evaluated, EPA will address the health-related questions regarding the ambient air concerns. If there is a health concern, it will be addressed as a separate operable unit in the remedial action phase.

Currently, the existing gas collection system installed between the landfill and the Riverside Gardens residential area, is working toward alleviating problems related to the migration of landfill-generated gases in the subsurface. Based on the present data available, it is recommended that a routine monitoring program be implemented for gas migration outside the collection system and in the Riverside Gardens area. If the existing gas collection system is repaired or a new system

constructed, proper maintenance will be required to prevent operational problems. Additionally, the ambient air should be monitored on the site to provide information on air quality and its potential effects.

Since "hot spot" soil areas were detected in the RI, it is recommended that these areas be covered during the remedial action phase. Also, it is recommended that cautionary signs be placed at the site to address direct contact with soil areas that may not have been identified as contaminated.

Table 10 provides a summary of the potential public health concerns resulting from the Endangerment Assessment for the Lees Lane Landfill Site. As shown in the table, there is no current evidence of an offsite problem related to the landfill site.

Future potential public health concerns are related to the elevated chromium levels in the groundwater at and upgradient of the site and to the potential release of methane and hazardous gases to the air and subsurface.

Since elevated chromium levels were detected in upgradient wells and no downgradient offsite impacts are evident, no remediation for the groundwater is recommended at this time. It is recommended that a monitoring program be implemented to establish baseline conditions at the site and to serve as an early warning system should site conditions change.

TABLE 10
PUBLIC HEALTH CONCERNS AND RECOMMENDATIONS
LEES LANE LANDFILL SITE
JEFFERSON COUNTY, KENTUCKY

Affected Area	Release Mechanism	Affected Media	Evidence of Current Public Health Concern	Potential Future Public Health Concern	Recommendations
Offsite	Leachate	Groundwater	No	Yes	Monitoring
	Runoff	Surface Water	No	No	None
		Sediments	No	No	None
		Surface Soil	No	No	None
	Gas Production	Gas Migration	No	Yes	Gas Collection System
		Air	No	Unknown	Monitoring
Onsite	Unrestricted Access	Surface Water	No	No	None
		Sediments	No	No	None
		Surface Soils	No	No	Cover "hot spots"
		Air	No	Unknown	Monitoring

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Appendix A
Chemical and Biological Toxicity of Lead

Pharmacokinetics

Kehoe (1961) showed that, in adults, eight percent of dietary lead is absorbed. Alexander et al.. (1973) showed that in children aged three months to 8.5 years, absorption was approximately 50 percent. Similar absorption characteristics were noted in rats by Forbes and Reina (1974).

Six and Goyer (1970 and 1972) observed that low dietary levels of calcium and iron and high fat levels increased the absorption of lead in laboratory animals. Ziegler (1978) observed a similar inverse relation between dietary calcium and lead absorption in human infants.

Once absorbed, lead is carried in blood as the dibasic phosphate or glycerophosphate and deposited in the skeleton as the poorly soluble tribasic phosphate. Barry (1975) showed that following long term exposure, 95 percent of the absorbed lead is deposited in the skeleton in adults and 72 percent in children. The remainder is localized in soft tissues, including the blood, where it is found primarily in the erythrocytes. Only when lead is present in relatively high amounts does a significant portion remain in the plasma.

In most species, including man, biliary excretion of lead predominates over urinary excretion (Cohen, 1970; Rabinowitz, et al., 1973).

Acute Effects

The effects of lead poisoning are detectable on the hematopoietic system at lower levels of exposure than any other organ or system. Piddington and White (1974) showed that lead interferes with heme synthesis by one of two actions. It may act by inhibiting heme synthetase, the enzyme that incorporates iron into protoporphyrin IX (a precursor of heme), or lead may block the entry of iron into the mitochondrion. In either case, heme production is decreased, leading to decreased hemoglobin production. A decrement in hemoglobin production is detectable at blood lead (referred to as PbB) levels of 50 ug/dl in adults (Tola et al., 1973) and 40 ug/dl in children (Betts et al., 1973).

Chronic Effects

Renal injury is most often observed after high level exposure to lead. Initial signs may include tubular damage, manifested by aminoaciduria, which was found by Clarkson and Kench (1956) to be uncommon at PbBs less than 70 ug/dl. Continued exposure may lead to decreased glomerular filtration, a progressive disease, found by Morgan et al., (1966) to occur at relatively low, 50 ug/dl, blood lead levels.

Lead may induce profound effects on the central nervous system. Landrigan and Baker (1976) showed that nerve conduction velocity slowed at PbBs of 50 ug/dl when no signs of neuromuscular involvement were apparent. Continued exposure may lead to lead palsy, manifested by muscle weakness, fatigue and paralysis. Usually the most active muscle groups are affected first. Degenerative changes occur in the motorneurons and their axons, affecting the upper limbs of adults and the lower limbs of children first. Subtle neurobehavioral effects appear to occur in the 40 to 80 ug/dl PbB range. More serious effects rarely occur at levels less than 80 ug/dl (Chisolm, 1968) and are usually higher, with a mean of 328 ug/dl reported in children (NAS, 1972). In children, exposure to lead occassionally produces progressive mental deterioration. Motor skills and speech slowly deteriorate and they may exhibit severe behavior disorders. The World Health Organization (WHO, 1977) noted the possibility of noticeable brain dysfunction in children at PbBs of 50 ug/dl.

Reproductive/Teratogenic Effects

McClain and Becker (1975) injected lead intraperitoneally in rats and noted teratogenic and embryotoxic effects. High lead concentrations (250 mg/l) in water, when administered to pregnant rats caused delayed fetal development and fetal resorption but no teratologic effects (Kimmel et al., 1976). There are no references to teratologic effects in man; however, stillbirths and miscarriages were reported among women working in lead trades in the late 19th and early 20th centuries. Lancranjan et al., (1975) reported an increased incidence of teratospermia (abnormally-shaped sperm) among men working in lead storage battery factories.

Mutagenic Effects

There was no significant excess of chromasome damage in cultured leukocytes obtained from cows accidentally poisoned with a mixture of heavy metals; toxic levels of lead were detected in liver and kidneys (IARC, 1972).

Carcinogenic Effects

An epidemiologic study of the causes of death among people exposed to high levels of lead revealed no increase in malignant neoplasms (Selander and Cramer, 1970). Cooper (1978) studied causes of death among lead-exposed workers and concluded that there was no consistent association between cancer incidence and length of employ or exposure. Kang (1980) looked at the same data and concluded that there was a correlation between lead exposure and excess cancer mortality. Zollinger (1953) showed that lead induced renal tumors in rats receiving lead acetate subcutaneously for up to 9.5 months. Azar (1973) confirmed these studies and observed that males were more susceptible and that a dose-response relationship existed. Despite evidence that some forms of lead are carcinogenic in some experimental animals, the International Agency for Research on Cancer considers the evidence to be of dubious significance with regard to man (IARC, 1972).

Appendix B
Chemical and Biological Toxicity of Arsenic

Arsenic

Pharmacokinetics

Absorption of arsenic depends on its chemical form and physical characteristics. Generally, the more soluble compounds are absorbed more readily than the less soluble ones. Greater than 95 percent of inorganic trivalent arsenic taken orally is absorbed and less than 5 percent is found in the feces (Ray-Bettley and O'Shea, 1975). Mappes (1977) reported that insoluble triselinide, when taken orally, passes through the gastrointestinal tract with negligible absorption.

Absorbed arsenic undergoes in vivo transformation to dimethylarsinic acid which may be considered a detoxified form. Charbonneau et al., (1978a,b) showed that in dogs, both erythrocytes and the liver are involved in the dimethylation and transport of the dimethylated forms. Braman and Foreback (1973) analyzed the arsenic in urine of four human volunteers. Two-thirds of the total arsenic was dimethylarsinic acid and 17 percent was pentavalent inorganic arsenic. Trivalent inorganic arsenic and methylarsonic acid each were present at 8 percent.

Mappes (1977) showed that 69 to 72 percent of the daily intake of arsenic in a human volunteer appeared in the urine. Most stored arsenic is found in skin, hair, teeth, bone, and nails. Of soft tissues, Kadowaki (1960) found that in man, the heart, kidney, liver, and lung tissues contained the highest levels of arsenic. Brain tissues had levels somewhat below other soft tissues.

Acute Effects

Webb (1966) showed that at least 78 enzymes from a wide variety of species are inhibited or inactivated by trivalent arsenic. The mechanism appears to be through the formation of arsenic-sulfide bonds, especially in oxidative enzymes involved in mitochondrial respiration.

Fowler et al (1977) demonstrated that pentavalent arsenate interferes with phosphate transport and phosphorylation. A competitive replacement of inorganic phosphate by arsenate causes an uncoupling of the mitochondrial oxidative phosphorylation.

Clinically, acute arsenic poisoning due to ingestion causes gastrointestinal disturbances, throat constriction, difficulty in swallowing, violent abdominal pain, vomiting, and diarrhea (Buchanan, 1962). Severe hypotension reflecting damage to the muscular system and death from cardiac failure may result. Trivalent arsenic is regarded as more acutely toxic than the pentavalent form with an estimated human lethal dose of 70 to 180 mg (Vallee et al., 1960).

Chronic Effects

Long term arsenic poisoning may affect the peripheral nervous system, the hematopoietic, cardiovascular, hepatic, and integumentary systems. Clinically, symptoms may include numbness and tingling of extremities, bone marrow injury, commonly aplastic anemia, myocardial toxicity including ventricular fibrillation, renal failure, copperlike pigmentation of the eyes; and the development of white bands across nails of hands and feet.

Teratogenic/Reproductive Effects

Golden hampsters, when injected with sodium arsenate produced offspring with a range of developmental malformations including anencephaly, rib malformations, and cleft lip and palate (Ferm et al., 1971). Similar results were noted by Hood et al., (1977) in mice receiving sodium arsenate via both oral and intraperitoneal routes.

Mutagenic Effects

Petres and Hundeiker (1968) and Petres et al., (1970) reported chromasome breakage in human leukocyte cultures after short term in vitro exposure to sodium arsenate in cultures obtained after long term exposure to arsenical compounds in vivo. The National Academy of Science (1977a) concluded that arsenic compounds have caused chromasome damage in a number of biological systems suggestive of a possible role in chemically induced mutagenesis.

Carcinogenic Effects

Tseng (1968) found a dose-response relationship between exposure to arsenic via drinking water and skin cancer prevalence in Taiwan. Similar health responses were reported in Chile (Borgono and Greiber, 1972), Argentina (Bergoglio, 1964), and Germany (Denk et al., 1969). The Taiwan experience was used by EPA to estimate lifetime cancer risk levels. Assuming a lifetime exposure occurring from the consumption of two liters of water and 6.5 grams of fish and shellfish per day, a level of 2,200 ug/l arsenic corresponds to the 10^{-6} risk level. Meaning, drinking water and eating seafood grown in water contaminated with arsenic at 2,200 ug/l for a lifetime increases one's risk of acquiring cancer to one additional case in a population of one million exposed individuals.

Appendix C
Chemical and Biological Toxicity of Benzene

Benzene

Pharmacokinetics

Inhalation is the most frequent route of benzene exposure in man. Toxic effects in humans have in the past been attributed to a combined inhalation and dermal exposure, although studies by Conca and Maltagliati (1955) with humans failed to prove that benzene was being absorbed percutaneously. Benzene primarily accumulates in the fatty tissue and bone marrow. The blood, liver, and kidney also contain significant amounts of benzene with the spleen, lungs, and brain containing lesser quantities.

The biotransformation of benzene occurs in the liver. Lutz and Schlatter (1977) showed that the metabolites of benzene appear to be bound covalently to residual protein of liver, brain, kidney, spleen, and fat in mice. The degree of binding is dose dependent and increases in both liver and bone marrow upon repeated exposure. Benzene appears to bind covalently with DNA in liver nuclei. This offers a model for the study of the mechanism of benzene toxicity and/or carcinogenesis in bone marrow.

Exhalation is the major route of excretion of unchanged benzene. Benzene toxicity in humans is usually caused by the inhalation of ambient air containing benzene vapors. Following cessation of exposure, the body burden is decreased either by exhaling benzene or by metabolism.

Acute Toxicity

A single exposure concentration of 66,000 mg/m^3 (20,000 ppm) commercial benzene has been reported to be fatal in man within 5 to 10 minutes[19]. Acute benzene poisoning is characterized by nausea, vomiting, irregular heart beat, dissiness, headache, and loss of consciousness[1]. Death is usually a result of cardiovascular collapse. In cases of acute poisoning, inflammation of the respiratory tract, hemorrhages of the lungs, congestion of the kidneys, and cerebral edema have also been observed. The TLV-TWA for benzene is presently 10 ppm with a STEL of 25 ppm (ACGIH, 1984).

Chronic Toxicity

Benzene is a known hematoxin. In man, it is casually related to pancytopemia and acute mycloblastic leukemia. Pancytopenia is a decrease in the major circulating formed elements in the blood: erythrocytes, leukocytes, and thrombocytes.

Aplastic anemia may be related to the use of benzene-containing adhesives in the shoe making industry. Aksoy, et al., (1967b, 1971) showed that the incidence of aplastic anemia declined following replacement of the adhesive with a benzene-free substance. Benzene levels in air to which the workers were exposed were in the range of 150 to 650 ppm.

A study of printing workers exposed to benzene in concentrations ranging from 71 to 1,060 ppm for three to five years revealed a significant incidence of hematological abnormalities. These included anemia, macrocytosis, and thrombocytopemia. Recovery from these hematological disorders was demonstrated following substitution of benzene with other solvents (IARC, 1981).

Doskin (1971) showed that the effects in occupationally-exposed groups to relatively low concentrations of benzene (10 to 40 ppm) for less than a year produced mild hematological effects. Mild thrombocytopenia was the most common abnormality observed with mild anemia. Also reported were lymphocytosis and bone marrow hypercellularity.

Altered immune function has been reported in both man and animal. Studies of workers exposed to benzene, but not seriously intoxicated by benzene, showed a decrease in complement levels of IgG and IgA. Studies on mice have demonstrated that the administration of benzene inhibits the function of B- and T- lymphocytes, while levels of IgM increased. This may explain why many benzene-intoxicated individuals readily succumb to infection[14].

Synergism and/or Antagonism

The interaction of benzene with other solvents, such as toluene, alters the rate of benzene metabolism. Benzene toxicity is prevented by the co-administration of

toluene, which inhibits benzene metabolism. This is because benzene and toluene are oxidized by many of the same hepatic enzyme systems (EPA, 1980).

Mutagenicity

In a study by Simon, et al., (1977), benzene did not show mutagenic activity in the Salmonella typhimusium/microsome in vitro assay. However, it has shown mutagenic activity in man and animals.

Benzene is a mitotic poison, producing a reduction in DNA sythesis in animal bone marrow cells in vitro and in cultured human cells. A significant increase in chromosomal aberrations in blood, bone marrow, and in the lymphocytes has been demonstrated by Doskin (1971) in humans and animals exposed to benzene at levels that caused hemotoxicity. Such abnormalities will persist long after the cessation of benzene exposure. It is not known whether occupational exposure to benzene at levels not producing overt hematological effects is capable of causing chromosomal abnormalities.

Teratogenicity

Some organic solvents (cyclohexane, carbon tetrachloride, chloroethane, and toluene) have been shown to produce congenital malformations in experimental animals (Casarett and Doull, 1980). The reported pancytopemia seen in workers exposed to toxic levels suggest the possibility that benzene could adversely affect the cells of a developing embryo. Pregnant mice that were administered benzene subcutaneously (3 mg/kg body weight) on day 13 of gestation produced fetuses with cleft palate, agnathia, and micrognathia. No other external visible defects were found in this study by Watanabe and Yoshida (1970).

Inhalation studies performed on animal models have proved too inconclusive to either refute or confirm that benzene is a teratogenic hazard. The effect of benzene on male and female fertility, preimplantation, parturition, and lactation also needs to be evaluated.

Carcinogencity

Doskin (1971) noted that animal data do not presently support conclusively the view that benzene is leukemogenic. This is probably due to the difference in metabolism between the animal model and man. Despite negative results from animal data, the evidence that benzene is a leukemogen for man is overwhelming. Workers exposed to benzene vapors of 150 to 210 ppm for up to 28 years (mean exposure = 11 years) had a high incidence of leukemia and Hodgkins disease (IARC, 1981).

An epidemiological study conducted on workers exposed to benzene in the manufacturing of a rubber product demonstrated convincingly an etiological implication of benzene as a leukemogen. A statistically significant (P 0.002) excess of leukemia was found in this cohort when compared with two control groups: the general American population and another industry not using benzene. There was a five-fold increased risk of leukemias and a ten-fold increased risk of myelocytic and monocytic leukemias combined. The environment of the workers was not contaminated with any other solvent other than benzene (IARC, 1981).

Also reported in association with benzene exposure have been lymphosarcoma, reticulum cell sarcoma, and multiple myeloma.

Appendix D
Chemical and Biological Toxicity of Chromium

Chromium

Pharmacokinetics

The analysis of the movement of chromium through various body pools and the determination of the turnover rates of these pools are complicated by several factors. Firstly, different chromium compounds will exhibit different kinetic characteristics in the body. Additionally, one chemical species of chromium can be transformed into another in the body, for instance, as by the reduction of Cr (VI) to Cr (III). Chromium, like other metals, circulates in the plasma primarily in a bound, nondiffusable form (Mertz, 1969). The high affinity of Cr (III) for the iron-binding protein sideophilin reflects the fact that this protein provides the normal mechanism of chromium transport to the tissues.

The half-life of plasma chromium is relatively short, and cells tend to accumulate the metal to levels higher than that present in the plasma. This accumulation results from the trapping of chromium compounds which penetrate the cells in the hexavalent form and then react with cell constituents. Inside the cell, the Cr (VI) will be reduced to Cr (III). In man, the highest concentrations of chromium are found in the lungs. The pulmonary levels tend to rise with age as the chromium content of other tissues fall. The lung obtains most of its chromium from the air, not from oral loads. Pulmonary chromium does not come into equilibrium with other body pools.

The majority of chromium is excreted in the urine.

Acute Effect

Changes in pulmonary dynamics have been observed among chromium electroplating workers (Bovett et al., 1977). The major effects of chromium on respiratory passages consist of ulceration of the nasal septum with subsequent perforation, and chronic rhinitis and pharyngitis. Forty-three to 85 percent of chromate plant workers exposed to concentrations of hexavalent chromium of 1 mg/m^3 were observed to have nasal septal perforations. A level of 68 ug/m^3 Cr (VI) was found to cause irritation to the eyes and throats of workers in a chromate

producing plant. Other symptoms of acute exposures to dust or mist include: coughing and wheezing, headache, eyspnea, fever, and weight loss. Tracheobronchial irritation and edema persist after other symptoms subside (Sittig, 1985). The present TLV-TWA for chromium metal is 0.5 mg/m^3.

The effects of chromium compounds on the skin have been recognized for over a century. The two major effects are the ulcerative changes of the skin developing from contact with various compounds of Cr (VI) and the development of allergic contact dematitis.

Chromium is an essential element for sugar and fat metabolism. The effects of exposure to low levels may be beneficial in deficiency states. The amounts necessary to cause toxic effects are much higher than those to correct deficiencies. The LD_{50} for Cr (III) intravenously is 10 mg/kg body weight exceeds by at least 4 orders of magnitude the dose needed to relieve impairment of glucose tolerance in chromium-deficient rats (EPA, 1978).

Chronic Effect

Dogs exposed to chromium (in the form of K_2CrO_4) for four years at a level of 0.45 mg/l in drinking water increased the chromium concentrations in the liver and spleen. However, no significant pathological changes occurred with such exposure in man. A concentration of 0.45 mg/l did not lead to any overt effects in four cases of prolonged human exposure (Anwar et al., 1961).

Mutagenicity

Soluble chromates of sodium, potassium and calcium stimulated mutagenesis in E. coli (Venitt and Levy, 1974).

Recently, it had been demonstrated that compounds of chromium possess the ability to cause clastogenesis and mutations. Both Cr III (as $CrCl_3$) and Cr (VI) (as $K_2Cr_2O_7$) produced morphologic changes in tertiary cultures of mouse fetal cells (Rafetto et al., 1977). Interestingly, hexavalent chromium caused more extensive

chromosomal aberrations than did trivalent chromium. The transformation frequency of similar adenovirus in Syrian hamster cells was elevated by calcium chromate.

There is little doubt that chromates can cause mutations and cell transformations. However, in the presence of liver enzymes or gastric juice (but not lung enzymes) chromates lose their mutagenic activity.

Teratogenicity

Sufficient data does not exist to make an evaluation of teratogencity.

Carcinogenicity

Rats injected intramuscularly once weekly for 20 weeks with calcium chromate (total dose 19 mg) developed a significant number of invasive spindle cell and pleomorphic cells at the injection site, none of which metastisized. The mean time of tumor appearance was 323 days. No tumors developed in control groups (Roe and Carter, 1969).

Mice receiving 5 ppm chromic acetate in drinking water for life did not show more tumors than control mice. Rats receiving a 5 ppm level of chromic acetate given in drinking water until death did not significantly increase the incidence of tumors.

The only well documented evidence of cancers associated with chromium exposure in humans involves the lungs. Industrial exposure vastly exceeds that attributed to food, water and air under normal conditions. Chromate workers who were followed over a period of 24 years had an excess lung cancer rate of 8.5. Excess incidence of all other cancers was only 1.3 (Taylor, 1966).

Appendix E

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